35/15

CENTERS FOR DISEASE CONTROL

September 26, 1986 / Vol. 35 / No. 1S

MMWR

MORBIDITY AND MORTALITY WEEKLY REPORT

Supplement

NEWARK PUBLIC LIBRARY

OCT 27 1986

U. S. GOVT. PUBS
DEPOSITORY 326

NIOSH
Recommendations for Occupational Safety and Health Standards

September 1986

U.S. DEPARTMENT OF HEALTH AND HUMAN SERVICES
Public Health Service
National Institute for Occupational Safety and Health
Centers for Disease Control
Atlanta, Georgia 30333

HE 20.7009:

READEX FICHE best copy available

INTRODUCTION

Acting under the authority of the Occupational Safety and Health Act of 1970 (Public Law 91-596), the National Institute for Occupational Safety and Health (NIOSH) develops, and periodically revises, recommendations for limits of exposure to potentially hazardous substances or conditions in the workplace. It also recommends preventive measures designed to reduce or eliminate adverse health effects associated with these hazards. In formulating these recommendations, NIOSH evaluates all known and available scientific information relevant to the potential hazard. These recommendations are then published and transmitted to the Department of Labor, Occupational Safety and Health Administration (OSHA) or Mine Safety and Health Administration (MSHA) for use in promulgating legal standards.

NIOSH recommendations are published in a variety of documents. Criteria Documents specify a NIOSH Recommended Exposure Limit (REL) and appropriate preventive measures designed to reduce or eliminate adverse health effects. Special Hazard Reviews, Occupational Hazard Assessments, and Technical Guidelines are other types of documents published by NIOSH to complement the Institute's recommendations for standards. These documents provide assessments, from a safety and health standpoint, of specific problems associated with a given agent or hazard and recommend appropriate control and surveillance methods.

Although these documents do not supplant the more comprehensive Criteria Document, they are prepared in such a way as to assist OSHA in the formulation of regulations. NIOSH also periodically presents testimony before various Congressional committees and at regulatory hearings convened by OSHA. The testimony presented always includes the current NIOSH policy concerning the particular hazard in question.

NIOSH also publishes documents known as Current Intelligence Bulletins (CIB) which review and evaluate emerging information on occupational hazards. These bulletins are based on the rapid evaluation of new and changing information on a particular hazard in light of existing knowledge.

The "NIOSH Recommendations for Occupational Safety and Health Standards" are based on existing NIOSH policy as previously published in any of the forms listed above. The intent of this table is to provide, in rapid-reference form, the most recent NIOSH REL or other recommendation for each potential hazard. The current OSHA Permissible Exposure Limit (PEL) or standard is also presented. Unless otherwise noted in the table, the NIOSH recommendations were originally published in Criteria Documents.

Note to Readers:

Copies of NIOSH publications are generally available from the U.S. Government Printing Office and the National Technical Information Service. Single copies of these publications may be obtained (while the supply lasts) from:

Publications Dissemination
Division of Standards Development
and Technology Transfer
National Institute for Occupational Safety and Health
4676 Columbia Parkway
Cincinnati, Ohio 45226
(513) 841-4287

Please enclose a self-addressed mailing label with your request.

Definitions of abbreviations and terms used in this publication:

Action level	the exposure concentration at which certain provisions of the NIOSH recommended standard must be initiated, such as periodic measurements of worker exposure, training of workers, and medical surveillance
Ca	NIOSH recommends that the substance be treated as a potential human carcinogen
CFR	Code of Federal Regulations
CIB	Current Intelligence Bulletin
dBA	decibel, weighted according to the A scale, which approximates the response of the human ear
ECG	electrocardiogram
J/cm^2	joules per square centimeter
μm	micrometer
μg/m^3	micrograms per cubic meter
mg/m^3	milligrams per cubic meter
mppcf	millions of particles per cubic foot
mW/cm^2	milliwatts per square centimeter
nm	nanometer
NIOSH	National Institute for Occupational Safety and Health
OSHA	Occupational Safety and Health Administration
PCB's	polychlorinated biphenyls
PCDD's	polychlorinated dibenzo-*p*-dioxins
PCDF's	polychlorinated dibenzofurans
PEL	Permissible Exposure Limit (OSHA)
ppb	parts per billion
ppm	parts per million
REL	Recommended Exposure Limit (NIOSH)
(Skin)	potential contribution to overall exposure by the cutaneous route including mucous membranes and eyes
TCDD	2,3,7,8-tetrachlorodibenzo-*p*-dioxin
TWA	time-weighted average

SUMMARY OF OSHA REGULATIONS AND NIOSH RECOMMENDATIONS FOR OCCUPATIONAL SAFETY AND HEALTH STANDARDS, 1986

		NIOSH		
Potential Hazard*	OSHA PEL'S/ Standard	REL'S[†]/Other Recommendations	Health Effect(s) Considered[§]	Comments
2-Acetylaminofluorene (NIOSH testimony at OSHA hearing September 1973)	No PEL Cancer-suspect agent Stringent workplace controls, recordkeeping, and medical surveillance required 29 CFR 1910.1014	Ca Use 29 CFR 1910.1014	Potential for cancer in humans; produced tumors of the liver, bladder, lungs, pancreas, and skin in animals	None
Acetylene (July 1976)	2,500 ppm (10% of lower explosive limit)	No exposure > 2,500 ppm ($2,662 mg/m^3$)	Asphyxia	Employers to check for and inform workers of contaminants such as arsine and phosphine
Acrylamide (October 1976)	$0.3 mg/m^3$, 8-hr TWA (Skin)	$0.3 mg/m^3$ TWA	Skin, eye, and nervous system effects	Prevent skin and eye contact
Acrylonitrile (January 1978; revised March 1978 as part of NIOSH testimony at OSHA hearing)	2 ppm, 8-hr TWA; 10 ppm ceiling (15 min) (Skin) 29 CFR 1910.1045	Ca 1 ppm, 8-hr TWA; 10 ppm ceiling (15 min) (Skin)	Brain tumors; lung and bowel cancer	Chest x ray required; first-aid and medical kits to be available during use; prevent skin contact
Aldrin/dieldrin (Special Hazard Review September 1978)	$0.25 mg/m^3$, 8-hr TWA (Skin)	Ca Lowest reliably detectable level	Potential for cancer in humans; produced tumors of the lungs, liver, thyroid, and adrenal glands in animals	Aldrin/dieldrin no longer produced in U.S.; prevent skin contact

---

*Date recommendation was published or testimony presented is in parentheses.

[†] NIOSH TWA recommendations are based on exposures up to 10 hours unless otherwise noted.

[§] Unless otherwise noted health effects cited are for humans.

		NIOSH		
Potential Hazard*	OSHA PEL'S/ Standard	REL'S[†]/Other Recommendations	Health Effect(s) Considered[§]	Comments
Alkanes (C5-C8) (March 1977)	All are 8-hr TWA values: Pentane: 1,000 ppm (2,950 mg/m^3); n-hexane: 500 ppm (1,800 mg/m^3); n-heptane: 500 ppm (2,000 mg/m^3); octane: 500 ppm (2,350 mg/m^3)	All are TWA values: Pentane: 120 ppm (350 mg/m^3); hexane: 100 ppm (350 mg/m^3); heptane: 85 ppm (350 mg/m^3); octane: 75 ppm (350 mg/m^3); Mixtures not to exceed 350 mg/m^3 TWA; All are ceiling values (15 min) singly or mixtures: pentane: 610 ppm (1,800 mg/m^3); hexane: 510 ppm (1,800 mg/m^3); heptane: 440 ppm (1,800 mg/m^3); octane: 385 ppm (1,800 mg/m^3); Action level set at 200 mg/m^3 for these substances	Skin and nervous system effects	None
Allyl chloride (September 1976)	1 ppm (3 mg/m^3), 8-hr TWA	1 ppm (3.1 mg/m^3) TWA; 3 ppm (9.3 mg/m^3) ceiling (15 min)	Liver, kidney, and lung effects	Urine, blood, and pulmonary function testing required
4-Aminodiphenyl (NIOSH testimony at OSHA hearing September 1973)	No PEL Cancer-suspect agent 29 CFR 1910.1011 Stringent workplace controls, recordkeeping, and medical surveillance required	Ca Use 29 CFR 1910.1011	Bladder cancer	None
Ammonia (July 1974)	50 ppm (35 mg/m^3), 8-hr TWA	50 ppm (34.8 mg/m^3) ceiling (5 min)	Respiratory irritation	Prevent eye contact
Anesthetic gases (see Waste anesthetic gases)				

Animal rendering processes (Occupational Hazard Assessment March 1981)	OSHA PEL's or NIOSH REL's for specific hazards are applicable		Mechanical injury; burns; heat stress; infections from biologic agents; chemical hazards	Guidelines for engineering controls and work practices to reduce injury and illness presented
Antimony (September 1978)	0.5 mg Sb/m^3, 8-hr TWA	0.5 mg Sb/m^3 TWA	Irritation; cardiovascular and lung effects	Chest x ray, pulmonary function testing, and electrocardiogram required
Arsenic, inorganic (September 1974; revised June 1975; reaffirmed July 1982 as part of NIOSH testimony at OSHA hearing)	10 μg As/m^3, 8-hr TWA 29 CFR 1910.1018	Ca 2 μg As/m^3 ceiling (15 min)	Lung and lymphatic cancer; dermatitis	Chest x ray required
Arsine (CIB August 1979)	0.2 mg/m^3 (0.05 ppm), 8-hr TWA	Ca 2 μg As/m^3 (0.002 mg As/m^3) ceiling (15 min)	Sudden extensive hemolysis	Workers to be warned of working with arsenic compounds in presence of freshly formed hydrogen
Asbestos (January 1972; revised December 1976; revised March 1984 as part of NIOSH testimony at Congressional hearing;	200,000 fibers/m^3, over 5 μm in length, 8-hr TWA; Action level of 100,000 fibers/m^3, 8-hr TWA 29 CFR 1910.1001	Ca 100,000 fibers/m^3, over 5 μm in length, 8-hr TWA in a 400-liter air sample	Lung cancer; mesothelioma; asbestosis	Chest x ray and pulmonary function testing required

[Continued on next page]

---

*Date recommendation was published or testimony presented is in parentheses.

[†] NIOSH TWA recommendations are based on exposures up to 10 hours unless otherwise noted.

[§] Unless otherwise noted health effects cited are for humans.

		NIOSH		
Potential Hazard*	OSHA PEL'S/ Standard	REL'S[†]/Other Recommendations	Health Effect(s) Considered[§]	Comments
reaffirmed June 1984 as NIOSH testimony at OSHA hearing)				
Asphalt fumes (September 1977)	None	5 mg/m^3 ceiling, measured as total particulate (15 min)	Eye and respiratory irritation	Medical surveillance required; prevent skin contact
Benzene (July 1974; revised August 1976; revised July 1977 as part of NIOSH testimony at OSHA hearing; revised March 1986 as part of NIOSH testimony at OSHA hearing)	10 ppm, 8-hr TWA; 25 ppm acceptable ceiling; 50 ppm maximum ceiling (10 min)	Ca 0.1 ppm (0.32 mg/m^3) 8-hr TWA; 1 ppm (3.2 mg/m^3) ceiling (15 min)	Cancer (leukemia)	Prevent skin contact
Benzidine (NIOSH testimony at OSHA hearing September 1973)	No PEL Cancer-suspect agent 29 CFR 1910.1010 Stringent workplace controls, recordkeeping, and medical surveillance required	Ca Use 29 CFR 1910.1010	Bladder, liver, and kidney cancer	None

Benzidine-based dyes (Special Hazard Review January 1980; revised in "Preventing health hazards from . . . benzidine congener dyes" January 1983)	No PEL for benzidine-based dyes	Ca Reduce exposure to lowest feasible level; replace with less toxic materials		and medical surveillance required. Urine monitoring for benzidine suggested
Benzoyl peroxide (June 1977)	5 mg/m^3, 8-hr TWA	5 mg/m^3 TWA	Respiratory and eye irritation; skin effects	None
Benzyl chloride (August 1978)	5 mg/m^3 (1 ppm), 8-hr TWA	5 mg/m^3 ceiling (15 min)	Irritation; skin and eye effects	Chest x ray and pulmonary function testing required
Beryllium (June 1972; revised August 1977 as part of NIOSH testimony at OSHA hearing)	2 μg/m^3, 8-hr TWA; 5 μg/m^3 acceptable ceiling; 25 μg/m^3 maximum ceiling (30 min)	Ca Not to exceed 0.5 μg Be/m^3	Lung cancer	Pulmonary function testing and chest x ray required
Boron trifluoride (December 1976)	1 ppm (3 mg/m^3) ceiling	No exposure limit recommended due to the absence of a reliable monitoring method	Respiratory effects	Appropriate engineering and work-practice controls to reduce exposure to lowest feasible level; pulmonary function testing required
1,3-Butadiene (CIB February 1984)	1,000 ppm (2,200 mg/m^3), 8-hr TWA	Ca Reduce exposure to lowest feasible level	Hematopoietic cancer; teratogenicity; reproductive system effects	Appropriate engineering and work-practice controls; restrict access to areas where 1,3-butadiene is used

---

[*] Date recommendation was published or testimony presented is in parentheses.

[†] NIOSH TWA recommendations are based on exposures up to 10 hours unless otherwise noted.

[§] Unless otherwise noted health effects cited are for humans.

Potential Hazard*	OSHA PEL'S/ Standard	NIOSH REL'S†/Other Recommendations	NIOSH Health Effect(s) Considered§	NIOSH Comments
Cadmium (August 1976; revised in CIB September 1984)	Fume: 0.1 mg/m³, 8-hr TWA; 0.3 mg/m³ ceiling; dust: 0.2 mg/m³, 8-hr TWA; 0.6 mg/m³ ceiling	Ca Reduce exposure to lowest feasible level	Lung cancer, prostatic cancer, renal system effects	None
Carbaryl (September 1976)	5 mg/m³, 8-hr TWA	5 mg/m³ TWA	Central nervous system and reproductive system effects	Workers to be warned of possible effects on reproductive system and to have only minimum exposure during pregnancy; prevent skin and eye contact
Carbon black (September 1978)	3.5 mg/m³, 8-hr TWA	3.5 mg/m³ TWA; Ca 0.1 mg/m³ TWA in presence of polycyclic aromatic hydrocarbons	Lung, cardiovascular and skin effects; cancer of the lymphatic-bone marrow complex when exposed to carbon black in the presence of polycyclic aromatic hydrocarbons	Chest x rays, pulmonary function testing, and ECG required
Carbon dioxide (August 1976)	5,000 ppm (9,000 mg/m³), 8-hr TWA	10,000 ppm (18,000 mg/m³) TWA; 30,000 ppm (54,000 mg/m³) ceiling (10 min)	Respiratory effects	None
Carbon disulfide (May 1977)	20 ppm, 8-hr TWA; 30 ppm acceptable ceiling; 100 ppm maximum ceiling (30 min)	1 ppm (3 mg/m³) TWA; 10 ppm (30 mg/m³) ceiling (15 min)	Cardiovascular, central nervous system, and reproductive system effects	Workers to be advised of potential effects on reproductive system
Carbon monoxide (August 1972)	50 ppm (55 mg/m³), 8-hr TWA	35 ppm (40 mg/m³), 8-hr TWA; 200 ppm (229 mg/m³) ceiling (no defined time)	Cardiovascular effects	None

Carbon tetrachloride (December 1975; revised June 1976)	10 ppm, 8-hr TWA; 25 ppm acceptable ceiling; 200 ppm maximum ceiling (5 min in 4 hr)	Ca 2 ppm (12.6 mg/m^3) ceiling in a 45-liter sample (60 min)	Liver cancer	REL based on lower limit of detection at time of document publication
Chlorine (May 1976)	1 ppm (3 mg/m^3) ceiling	0.5 ppm (1.45 mg/m^3) ceiling (15 min)	Eye and respiratory irritation	Chest x rays required
Chloroethane (CIB August 1978)	1,000 ppm (2,600 mg/m^3), 8-hr TWA	To be handled in the workplace with caution	Central nervous system effects; possible liver and/or kidney effects	Exposures should be minimized due to the structural similiarity to the carcinogenic chloroethanes
Chloroform (September 1974; revised June 1976)	50 ppm (240 mg/m^3) ceiling	Ca 2 ppm (9.78 mg/m^3) ceiling in a 45-liter sample (60 min)	Liver or kidney tumors and central nervous system effects	None
bis-Chloromethyl ether (NIOSH testimony at OSHA hearing September 1973)	No PEL Cancer-suspect agent Stringent workplace controls, recordkeeping, and medical surveillance required 29 CFR 1910.1008	Ca Use 29 CFR 1910.1008	Lung cancer	None
Chloroprene (August 1977)	25 ppm (90 mg/m^3), 8-hr TWA	Ca 1 ppm (3.6 mg/m^3) ceiling (15 min)	Lung and skin cancer; reproductive effects	Chest x ray and pulmonary function testing required; pregnant workers to be counseled about continuing work with chloroprene
Chromic acid (July 1973; revised—see Chromium (VI) December 1975)	1 mg/10 m^3 (100 μg/m^3) ceiling	25 μg/m^3 (0.025 mg/m^3) TWA; 50 μg/m^3 (0.05 mg/m^3) ceiling (15 min) as noncarcinogenic Cr (VI)	Nasal ulceration	None

---

[*] Date recommendation was published or testimony presented is in parentheses.

[†] NIOSH TWA recommendations are based on exposures up to 10 hours unless otherwise noted.

[§] Unless otherwise noted health effects cited are for humans.

		NIOSH		
Potential Hazard*	**OSHA PEL'S/ Standard**	**REL'S[†]/Other Recommendations**	**Health Effect(s) Considered[§]**	**Comments**
Chromium (VI) (December 1975)	1 mg/10 m^3 (100 μg/m^3) ceiling	Ca Carcinogenic Cr (VI): 1 μg/m^3 TWA; other Cr (VI): 25 μg/m^3 TWA; 50 μg/m^3 ceiling (15 min)	Lung cancer, skin ulcers, and lung irritation	Employer must demonstrate absence of carcinogenic Cr (VI); x ray required
Chrysene (Special Hazard Review June 1978)	0.2 mg/m^3, 8-hr TWA	Ca To be controlled as an occupational carcinogen	Potential for cancer in humans; produced liver and skin tumors in animals	Document also contains control recommendations for polycyclic aromatic hydrocarbons
Coal gasification plants (September 1978)	OSHA PEL's or NIOSH REL's for specific hazards are applicable		Various effects depending on substances present; potential for skin cancer	Extensive work-practice and control procedures recommended
Coal liquefaction, volumes I and II (Occupational Hazard Assessment March 1981)	OSHA PEL's or NIOSH REL's for specific hazards are applicable		Various effects depending on substances present; potential for skin cancer	Extensive work-practice and control procedures recommended
Coal tar products (September 1977)	0.2 mg/m^3, 8-hr TWA (benzene-soluble fraction) 29 CFR 1910.1002 (coal tar pitch volatiles)	Ca 0.1 mg/m^3 TWA (cyclohexane-extractable fraction)	Lung and skin cancer	Includes coal tar, creosote, and coal-tar pitch; pulmonary function testing and chest x rays required

Cobalt (Occupational Hazard Assessment October 1981)	0.1 mg/m^3, 8-hr TWA	NIOSH has concluded that there is insufficient evidence to warrant recommending a new exposure limit	Dermatitis; potential for pulmonary fibrosis	Includes recommendations for engineering controls, work practices, protective equipment, worker education, monitoring, and medical surveillance
Coke oven emissions (February 1973; revised November 1975 as part of NIOSH testimony at OSHA hearing)	150 μg/m^3, 8-hr TWA 29 CFR 1910.1029	Ca 0.5-0.7 mg/m^3 (500-700 μg/m^3) (total particulates) as screening level	Lung cancer	Chest x ray required; work practices to minimize exposure to emissions
Confined spaces, working in (December 1979)	Covered under numerous OSHA regulations for General Industry (29 CFR 1910)	Various recommendations including a permit system to prevent worker injury and death	Injury and death	None
Cotton dust (September 1974; reaffirmed September 1983 as part of NIOSH testimony at OSHA hearing)	Yarn manufacturing: 200 μg/m^3, 8-hr TWA; slashing and weaving operations: 750 μg/m^3, 8-hr TWA; all other operations: 500 μg/m^3, 8-hr TWA 29 CFR 1910.1043	<200 μg/m^3 lint-free cotton dust	Pulmonary disease (byssinosis)	Pulmonary function testing required
Cresol (February 1978)	5 ppm (22 mg/m^3), 8-hr TWA (Skin)	2.3 ppm (10 mg/m^3) TWA	Skin, liver, kidney, and pancreas effects	Applies to mixtures of cresols and cresylic acid; prevent skin and eye contact; possible delayed effects

---

[*] Date recommendation was published or testimony presented is in parentheses.

[†] NIOSH TWA recommendations are based on exposures up to 10 hours unless otherwise noted.

[§] Unless otherwise noted health effects cited are for humans.

		NIOSH		
Potential Hazard*	OSHA PEL'S/ Standard	REL'S[†]/Other Recommendations	Health Effect(s) Considered[§]	Comments
DDT (Special Hazard Review September 1978)	1 mg/m^3, 8-hr TWA (Skin)	Ca Lowest reliably detectable level; 0.5 mg/m^3 TWA by NIOSH-validated method	Potential for cancer in humans; produced tumors of the liver, lungs, and lymphatic system in animals	Prevent skin contact
2,4-Diaminoanisole and its salts (CIB January 1978)	None	Ca Reduce exposure to lowest feasible level	Potential for cancer in humans; produced tumors of the thyroid, skin, and lymphatic system in animals	Prevent skin contact; engineering and work-practice controls are recommended
o-Dianisidine-based dyes (Joint NIOSH/OSHA Health Hazard Alert December 1980)	None	Ca Should be handled in the workplace with caution; exposures should be minimized	Potential for cancer in humans; produced tumors of the bladder, stomach, and mammary glands in animals	Substitute less toxic dyes wherever possible
Dibromochloropropane (January 1978)	1 ppb, 8-hr TWA; eye and skin contact to be avoided 29 CFR 1910.1044	10 ppb (0.1 mg/m^3) TWA (NIOSH recommendation superseded by OSHA standard promulgated in 1978)	Sterility; renal and liver effects	Regulated by OSHA as a carcinogen
3,3'-Dichlorobenzidine (NIOSH testimony at OSHA hearing September 1973)	No PEL Cancer-suspect agent Stringent workplace controls, recordkeeping, and medical surveillance required 29 CFR 1910.1007	Ca Use 29 CFR 1910.1007	Potential for cancer in humans; produced tumors of the liver, bladder, and lungs in animals	None

1,1-Dichloroethane (CIB August 1978)	100 ppm (400 mg/m^3), 8-hr TWA	To be handled in the workplace with caution	Central nervous system effects; possible liver and/or kidney damage	Exposures should be minimized due to the structural similiarity to the carcinogenic chloroethanes
Dieldrin (see Aldrin/dieldrin)				
Di-2-Ethylhexyl Phthalate (DEHP)(Special Hazard Review March 1983)	5 mg/m^3, 8-hr TWA	Ca Reduce exposure to lowest feasible level	Potential for cancer in humans; produced liver tumors in animals	DEHP, widely used in the quantitative fit testing of respirators, should be replaced with less toxic material such as refined corn oil
Diisocyanates (September 1978)	Toluene diisocyanate (TDI): 0.02 ppm (0.14 mg/m^3) ceiling; diphenylmethane diisocyanate (MDI): 0.02 ppm (0.2 mg/m^3) ceiling	All values given in μg/m^3 and all ceiling values for 10 min (each equivalent to 5 ppb TWA and 20 ppb ceiling): TDI: 35 TWA, 140 ceiling; MDI: 50 TWA, 200 ceiling; hexamethylene diisocyanate (HDI): 35 TWA, 140 ceiling; naphthalene diisocyanate (NDI): 40 TWA, 170 ceiling; isophorone diisocyanate (IPDI): 45 TWA, 180 ceiling; dicyclohexylmethane-4,4′-diisocyanate (hydrogenated MDI): 55 TWA, 210 ceiling; other diisocyanates to be controlled to 20 ppb ceiling and 5 ppb TWA	Respiratory effects and sensitization; pulmonary irritation	Chest x ray and pulmonary function testing required

---

[*] Date recommendation was published or testimony presented is in parentheses.

[†] NIOSH TWA recommendations are based on exposures up to 10 hours unless otherwise noted.

[§] Unless otherwise noted health effects cited are for humans.

		NIOSH		
Potential Hazard[*]	**OSHA PEL'S/ Standard**	**REL'S**[†]**/Other Recommendations**	**Health Effect(s) Considered**[§]	**Comments**
4-Dimethylamino-azobenzene (NIOSH testimony at OSHA hearing September 1973)	No PEL Cancer-suspect agent Stringent workplace controls, recordkeeping, and medical surveillance required 29 CFR 1910.1015	Ca Use 29 CFR 1910.1015	Potential for cancer in humans; produced tumors of the liver and bladder in animals	None
Dinitro-ortho-cresol (February 1978)	0.2 mg/m^3, 8-hr TWA (Skin)	0.2 mg/m^3 TWA	Central nervous system and metabolic effects	Blood and urine monitoring required; prevent skin and eye contact; possible delayed effects
Dinitro-toluenes (CIB July 1985)	1.5 mg/m^3, 8-hr TWA (Skin)	Ca Reduce exposure to lowest feasible level	Potential for cancer in humans; produced tumors of the liver, skin, and kidneys in animals; reproductive system effects	Prevent skin contact
Dioxane (September 1977)	100 ppm (360 mg/m^3), 8-hr TWA (Skin)	Ca 1 ppm (3.6 mg/m^3) ceiling (30 min)	Potential for cancer in humans; produced tumors of liver, lungs, and nasal cavity in animals; effects on liver and kidney	Blood and urine testing required; prevent skin contact
Dioxin (see 2,3,7,8-Tetrachloro-dibenzo-*p*-dioxin)				
Elevated workstations, emergency egress from (December 1975)	Sections under Subpart E, Means of Egress, General Industry Standards, and Subpart R, Special Industries (29 CFR 1910.261)	Various recommendations concerning means and availability of egress	Trauma and injury	None

Epichlorohydrin (September 1976; revised in CIB October 1978)	5 ppm (19 mg/m^3), 8-hr TWA	Ca Occupational exposure to epichlorohydrin to be minimized	Respiratory cancer; mutagenesis; reproductive effects; skin, kidney, liver, and respiratory effects	Prevent skin contact
2-Ethoxy-ethanol (see Glycol ethers)				
Ethyl chloride (see Chloroethane)				
Ethylene dibromide (August 1977; revised November 1983; reaffirmed February 1984 as part of NIOSH testimony at OSHA hearing)	20 ppm, 8-hr TWA; 30 ppm acceptable ceiling; 50 ppm maximum peak (5 min)	Ca 0.045 ppm (0.38 mg/m^3), 8-hr TWA; 0.13 ppm (1 mg/m^3) ceiling (15 min)	Potential for cancer in humans; mutagenesis; damage to skin, eyes, cardiovascular, liver, spleen, reproductive, respiratory, and central nervous systems	Workers to be warned of potential for reproductive abnormalities and cancer; hazardous liquid; prevent skin contact
Ethylene dichloride (March 1976; revised in CIB April 1978; revised September 1978)	50 ppm, 8-hr TWA; 100 ppm acceptable ceiling; 200 ppm maximum ceiling (5 min in 3 hr)	Ca 1 ppm (4 mg/m^3) TWA; 2 ppm (8 mg/m^3) ceiling (15 min)	Potential for cancer in humans; nervous system, respiratory, cardiovascular, and liver effects	Nursing infants of exposed mothers at risk

---

[*] Date recommendation was published or testimony presented is in parentheses.

[†] NIOSH TWA recommendations are based on exposures up to 10 hours unless otherwise noted.

[§] Unless otherwise noted health effects cited are for humans.

Potential Hazard*	OSHA PEL'S/ Standard	NIOSH REL'S[†]/Other Recommendations	 Health Effect(s) Considered[§]	 Comments
Ethyleneimine (NIOSH testimony at OSHA hearing September 1973)	0.5 ppm (1 mg/m^3), 8-hr TWA (Skin) 29 CFR 1910.1012	Ca Use 29 CFR 1910.1012	Potential for cancer in humans; produced tumors of the liver and lung in animals	Stringent workplace controls and medical surveillance required
Ethylene oxide (Special Hazard Review September 1977; revised July 1983 as part of NIOSH testimony at OSHA hearing)	1 ppm (1.8 mg/m^3), 8-hr TWA 29 CFR 1910.1047	Ca 5 ppm (9 mg/m^3) ceiling (10 min/day); <0.1 ppm (0.18 mg/m^3), 8-hr TWA	Peritoneal cancer; leukemia; mutagenesis; reproductive effects	Blood monitoring and medical counseling recommended
Ethylene thiourea (Special Hazard Review October 1978)	None	Ca Should be used in encapsulated form in industry; worker exposure to be minimized	Potential for cancer and teratogenicity in humans; produced tumors of the liver, thyroid, and lymphatic system in animals	Workers to be informed of carcinogenic and teratogenic hazards; special attention to be given to thyroid function tests
Excavations, development of draft construction safety standards for (Technical Guideline May 1983)	Many aspects covered under OSHA regulations governing excavations, trenching, and shoring practices in the construction industry (29 CFR 1926, Subpart P)	Many work-practice recommendations concerning safety standards for excavations	Injury and death	None
Fibrous glass (April 1977)	Nuisance dust PEL applies, 15 mg/m^3 total dust; 5 mg/m^3 respirable fraction	3 million fibers/m^3 TWA (fibers $\leq$ 3.5 μm diameter and $\geq$ 10 μm length); 5 mg/m^3 TWA (total fibrous glass)	Eye, skin, and respiratory effects	NIOSH recommends that this REL also apply to other synthetic fibers

Fluorides, inorganic (June 1975)	2.5 mg F/m^3, 8-hr TWA	2.5 mg F/m^3 TWA	Kidney and bone effects	Urine monitoring required
Fluorocarbon polymers, decomposition products of (September 1977)	None	Various recommendations emphasizing good work practices, engineering controls, and medical surveillance	Lung effects; polymer fume fever	Workroom air to be monitored for inorganic fluorides and hydrogen fluoride
Formaldehyde (December 1976; revised in CIB April 1981; revised May 1986 as part of NIOSH testimony at OSHA hearing)	3 ppm, 8-hr TWA; 5 ppm acceptable ceiling; 10 ppm maximum ceiling (30 min)	Ca 0.1 ppm ceiling (15 min); represents the lowest reliably quantifiable concentration	Potential for cancer in humans; produced tumors of the nasal cavity in animals	Medical surveillance; skin protection
Foundries (September 1985)	Many aspects covered under the numerous OSHA regulations for general industry (29 CFR 1910)	Various recommendations emphasizing good work practices, engineering controls, and medical surveillance	Cancer; respiratory disease; heat-induced illness; noise-induced hearing loss; vibration-induced disorders; eye injuries; traumatic and ergonomic injuries	Recommendations limited to foundries that pour molten metal into sand molds
Furfuryl alcohol (March 1979)	50 ppm (200 mg/m^3), 8-hr TWA	50 ppm (200 mg/m^3) TWA	Respiratory effects	None

---

*Date recommendation was published or testimony presented is in parentheses.

†NIOSH TWA recommendations are based on exposures up to 10 hours unless otherwise noted.

§Unless otherwise noted health effects cited are for humans.

Potential Hazard*	OSHA PEL'S/ Standard	NIOSH		
		REL'S†/Other Recommendations	Health Effect(s) Considered§	Comments
Glycidyl ethers (June 1978; revised in CIB October 1978)	All values in ppm (mg/m^3): allyl glycidyl ether (AGE): 10 (45) ceiling; n-butyl glycidyl ether (BGE): 50 (270), 8-hr TWA; di-2,3-epoxypropyl ether (DGE): 0.5 (2.8), 8-hr TWA; isopropyl glycidyl ether (IGE): 50 (240), 8-hr TWA; phenyl glycidyl ether (PGE): 10 (60), 8-hr TWA	All are ceiling values (15 min) in ppm (mg/m^3): AGE: 9.6 (45); BGE: 4.4 (30); DGE: 0.2 (1) Ca; IGE: 50 (240); PGE: 1 (5)	DGE: Potential for cancer in humans; produced skin tumors in animals; DGE and other glycidyl ethers: skin and mucous membrane effects; sensitization potential; possible hematopoietic and reproductive system effects	Possible additive effects with mixtures; medical surveillance
Glycol ethers (CIB May 1983)	2-Methoxyethanol: 25 ppm (80 mg/m^3), 8-hr TWA (Skin); 2-ethoxyethanol: 200 ppm (740 mg/m^3), 8-hr TWA (Skin)	Reduce exposure to lowest feasible level	Reproductive effects; teratogenicity	Prevent skin contact
Grain elevators and feed mills, occupational safety in (Technical Guideline September 1983; reaffirmed June 1984 as part of NIOSH testimony at OSHA hearing)	Many general aspects (e.g., protective equipment, dust control, etc.) covered under the numerous OSHA regulations for general industry (29 CFR 1910)	Various recommendations for control of combustible dusts and ignition sources, machine guarding, isolation and lockouts, bin entry, training, and personal protective equipment	Injury and death	Health hazards from exposure to fumigants, pesticides, and grain dust
Hexachloroethane (CIB August 1978)	1 ppm (10 mg/m^3), 8-hr TWA (Skin)	Ca Reduce exposure to lowest feasible level	Potential for cancer in humans; produced liver tumors in animals	None

Hot environments (June 1972; revised April 1986)	None	Sliding scale limits based upon environmental and metabolic heat loads	Heat-induced illnesses	Recommendations include acclimatization, strict work practices, protective equipment, and medical surveillance
Hydrazines (June 1978)	All values in ppm (mg/m³): hydrazine: 1 (1.3), 8-hr TWA; 1,1-dimethylhydrazine: 0.5 (1.0), 8-hr TWA; phenylhydrazine: 5 (22), 8-hr TWA; methylhydrazine: 0.2 (0.35) ceiling	Ca All are ceiling values (120 min) in ppm (mg/m³): hydrazine: 0.03 (0.04); 1,1-dimethylhydrazine: 0.06 (0.15); phenylhydrazine: 0.14 (0.6); methylhydrazine: 0.04 (0.08)	Potential for cancer in humans; produced tumors of the lung, liver, blood vessels, and intestines in animals; blood, liver, and skin effects	Blood and urine monitoring and chest x ray required; bowel examination for workers over age 40
Hydrogen cyanide and cyanide salts (October 1976)	Hydrogen cyanide: 10 ppm (11 mg/m³), 8-hr TWA (Skin); cyanide: 5 mg CN/m³, 8-hr TWA (Skin)	4.7 ppm (5 mg CN/m³) ceiling (10 min)	Thyroid, blood, respiratory system effects	Concurrent measurement required for HCN when measuring for cyanide salt; trained first-aid personnel and first-aid kits to be available during use; prevent skin and eye contact
Hydrogen fluoride (March 1976)	3 ppm, 8-hr TWA	3 ppm (2.5 mg F/m³) TWA; 6 ppm (5.0 mg F/m³) ceiling (15 min)	Skin, eye, and airway irritation; bone effects	Pelvic x ray (male workers only) and urine testing required
Hydrogen sulfide (May 1977)	20 ppm acceptable ceiling; 50 ppm maximum ceiling (10 min)	10 ppm (15 mg/m³) ceiling (10 min)	Irritation; severe acute effects involving nervous and respiratory systems	Continuous monitoring required if potential exists for exposure to ≥ 70 mg/m³ (47 ppm); evacuation required at this level

---

[*] Date recommendation was published or testimony presented is in parentheses.

[†] NIOSH TWA recommendations are based on exposures up to 10 hours unless otherwise noted.

[§] Unless otherwise noted health effects cited are for humans.

		NIOSH		
Potential Hazard*	OSHA PEL'S/ Standard	REL'S†/Other Recommendations	Health Effect(s) Considered§	Comments
Hydroquinone (April 1978)	2 mg/m^3, 8-hr TWA	0.44 ppm (2 mg/m^3) ceiling (15 min)	Eye and skin effects	Special provisions for darkroom use
Identification system for occupationally hazardous materials (December 1974)	Sections of Hazard Communication (29 CFR 1910.1200) and carcinogen standards may be applicable	Complete designation system for occupationally hazardous materials	None	Includes definition, safety data sheets, alert symbols, and label statements
Isopropyl alcohol (March 1976)	400 ppm (980 mg/m^3), 8-hr TWA	400 ppm (984 mg/m^3) TWA; 800 ppm (1,968 mg/m^3) ceiling (15 min)	Mucous membrane irritation; possible cancer threat in manufacturing process	Stringent work practices and medical surveillance for manufacturing workers required
Kepone (January 1976)	None	Ca 1 μg/m^3 TWA	Liver cancer; nervous system effects	Liver function testing required
Ketones (June 1978)	All are 8-hr TWA values in ppm (mg/m^3): acetone: 1,000 (2,400); methyl ethyl ketone: 200 (590); methyl n-propyl ketone: 200 (700); methyl n-butyl ketone: 100 (410); methyl n-amyl ketone: 100 (465); methyl isobutyl ketone: 100 (410); methyl isoamyl ketone: none; diisobutyl ketone: 50 (290); cyclohexanone: 50 (200); mesityl oxide: 25 (100); diacetone alcohol: 50 (240); isophorone: 25 (140)	All are TWA values in ppm (mg/m^3): acetone: 250 (590); methyl ethyl ketone: 200 (590); methyl n-propyl ketone: 150 (530); methyl n-butyl ketone: 1 (4); methyl n-amyl ketone: 100 (465); methyl isobutyl ketone: 50 (200); methyl isoamyl ketone: 50 (230); diisobutyl ketone: 25 (140); cyclohexanone: 25 (100); mesityl oxide: 10 (40); diacetone alcohol: 50 (240); isophorone: 4 (23)	Irritation; liver, kidney, and nervous system effects	Urinalysis required; workers exposed to methyl n-butyl ketone to be warned of nervous system effects

Land-based oil and gas well drilling, comprehensive safety recommendations for (Technical Guideline September 1983; reaffirmed March 1984 as part of NIOSH testimony at OSHA hearing)	Many aspects covered under the numerous OSHA regulations for general industry (29 CFR 1910)	Various recommendations for safe work practices and technologic improvements	Injury and death	Many tasks, types of equipment, and conditions are not covered by existing regulations
Lead, inorganic (January 1973; revised May 1978)	50 µg Pb/m^3, 8-hr TWA; over 8-hr exposure to be determined by formula 29 CFR 1910.1025	< 100 µg Pb/m^3 TWA; air level to be maintained so that worker blood lead remains ≤ 60 µg/100g	Kidney, blood, and nervous system effects	Blood monitoring required
Lockout/tagout, guidelines for controlling hazardous energy during maintenance and servicing (Technical Guideline September 1983)	Many aspects covered under OSHA regulations for general industry (29 CFR 1910) and construction standards (29 CFR 1926)	Work-practice recommendations for controlling hazardous energy during maintenance and servicing activities	Injury and death	"Energy" defined in this document as kinetic energy, potential energy, electrical energy, and thermal energy
Logging from felling to first haul (July 1976)	None	Extensive work-practice and personal protection recommendations	Primarily trauma and falls	Tetanus toxoid inoculations and first-aid programs to be instituted
Malathion (June 1976)	15 mg/m^3, 8-hr TWA	15 mg/m^3 TWA	Nervous system effects	Prevent skin contact; blood monitoring required

---

[*] Date recommendation was published or testimony presented is in parentheses.

[†] NIOSH TWA recommendations are based on exposures up to 10 hours unless otherwise noted.

[§] Unless otherwise noted health effects cited are for humans.

Potential Hazard*	OSHA PEL'S/ Standard	NIOSH		
		REL'S†/Other Recommendations	Health Effect(s) Considered§	Comments
Mercury, inorganic (August 1973)	0.1 mg/m^3 acceptable ceiling	0.05 mg Hg/m^3, 8-hr TWA	Central nervous system and mental effects	Work practices, sanitation, monitoring, and medical surveillance emphasized
2-Methoxy-ethanol (see Glycol ethers)				
Methyl alcohol (March 1976)	200 ppm (260 mg/m^3), 8-hr TWA	200 ppm (262 mg/m^3) TWA; 800 ppm (1,048 mg/m^3) ceiling (15 min)	Blindness; metabolic acidosis	None
Methyl chloro-methyl ether (NIOSH testimony at OSHA hearing September 1973)	No PEL Cancer-suspect agent Stringent workplace controls, recordkeeping, and medical surveillance required 29 CFR 1910.1006	Ca Use 29 CFR 1910.1006	Lung cancer	None
4,4'-Methylenebis-(2-chloroaniline) (MOCA) (Special Hazard Review September 1978)	Standard formally revoked by OSHA, August 1975	Ca 3 μg/m^3 TWA (lowest detectable limit)	Potential for cancer in humans; produced liver and lung tumors in animals	Chest x ray; blood and urine testing required
Methylene chloride (March 1976; revised April 1986 in CIB)	500 ppm, 8-hr TWA; 1,000 ppm acceptable ceiling; 2,000 ppm acceptable maximum peak for 5 min in any 2-hr period above the acceptable ceiling for an 8-hr shift	Ca Reduce exposure to lowest feasible limit	Potential for cancer in humans; produced tumors of the lung, liver, salivary, and mammary glands in animals	None

4,4'-Methylene-dianiline (CIB July 1986)	None	Ca Reduce exposure to lowest feasible limit	Bladder cancer; skin and liver effects	Prevent skin contact
Methyl parathion (September 1976)	None	0.2 mg/m^3 TWA	Central nervous system effects	Prevent skin contact; blood monitoring required
Monohalo-methanes (CIB September 1984)	Methyl chloride: 100 ppm, 8-hr TWA; 200 ppm ceiling; 300 ppm acceptable maximum peak for 5 min in any 3-hr period above the acceptable ceiling for an 8-hr shift; methyl bromide: 20 ppm (80 mg/m^3) ceiling (Skin); methyl iodide: 5 ppm (28 mg/m^3), 8-hr TWA (Skin)	Ca Exposure to methyl chloride, methyl bromide, and methyl iodide should be reduced to lowest feasible level	Potential for cancer in humans; produced tumors of the kidney, forestomach, and lung in animals; methyl chloride should also be considered a potential teratogen	None
alpha-Naphthyl-amine (NIOSH testimony at OSHA hearing September 1973)	No PEL Cancer-suspect agent Stringent workplace controls, recordkeeping, and medical surveillance required 29 CFR 1910.1004	Ca Use 29 CFR 1910.1004	Bladder cancer	None
beta-Naphthyl-amine (NIOSH testimony at OSHA hearing September 1973)	No PEL Cancer-suspect agent Stringent workplace controls, recordkeeping, and medical surveillance required 29 CFR 1910.1009	Ca Use 29 CFR 1910.1009	Bladder cancer	None

---

[*] Date recommendation was published or testimony presented is in parentheses.

[†] NIOSH TWA recommendations are based on exposures up to 10 hours unless otherwise noted.

[§] Unless otherwise noted health effects cited are for humans.

Potential Hazard[*]	OSHA PEL'S/ Standard	NIOSH REL'S[†]/Other Recommendations	NIOSH Health Effect(s) Considered[§]	NIOSH Comments
Niax® Catalyst ESN (Joint NIOSH/ OSHA CIB May 1978)	OSHA and NIOSH recommend that exposure to Niax® Catalyst ESN and its components, dimethylaminopropionitrile and bis [2-(dimethylamino)ethyl] ether, be minimized		Urological disorders; nervous system effects	Work-practice and engineering controls to reduce exposure
Nickel carbonyl (Special Hazard Review May 1977)	1 ppb (7 $\mu g/m^3$), 8-hr TWA	Ca 1 ppb (7 $\mu g/m^3$) TWA (lowest detectable level)	Lung and nasal cancer	Chest x ray, pulmonary function testing, and urine monitoring required
Nickel, inorganic compounds (May 1977)	1 mg Ni/m^3, 8-hr TWA	Ca 15 μg Ni/m^3 TWA	Lung and nasal cancer; skin effects	Chest x ray and pulmonary function testing required
Nitric acid (March 1976)	2 ppm (5 mg/m^3), 8-hr TWA	2 ppm (5 mg/m^3) TWA	Dental erosion; nasal/lung irritation	Prevent skin and eye contact; chest x ray required
Nitriles (September 1978)	Acetonitrile: 40 ppm (70 mg/m^3), 8-hr TWA; tetramethyl succinonitrile: 0.5 ppm (3 mg/m^3), 8-hr TWA (Skin)	All are TWA values in ppm (mg/m^3): acetonitrile: 20 (34); n-butyronitrile: 8 (22); isobutyronitrile: 8 (22); propionitrile: 6 (14); malononitrile: 3 (8); adiponitrile: 4 (18); succinonitrile: 6 (20). All ceiling values (15 min) in ppm (mg/m^3): acetone cyanohydrin: 1 (4); glycolonitrile: 2 (5); tetramethyl succinonitrile: 1 (6). When present as mixtures or with other sources of cyanide, exposure to be considered additive and environmental limit to be calculated.	Hepatic, renal, respiratory, cardiovascular, gastrointestinal, and nervous system effects	Chest x ray and pulmonary function testing required; trained personnel and first-aid kits to be available during use; prevent skin and eye contact

4-Nitrobiphenyl (NIOSH testimony at OSHA hearing September 1973)	No PEL Cancer-suspect agent Stringent workplace controls, recordkeeping, and medical surveillance required 29 CFR 1910.1003	Ca Use 29 CFR 1910.1003	Potential for cancer in humans; produced bladder tumors in animals	None
Nitrogen, oxides of (March 1976)	NO_2: 5 ppm ($9\ mg/m^3$) ceiling; NO: 25 ppm ($30\ mg/m^3$), 8-hr TWA	NO_2: 1 ppm ($1.8\ mg/m^3$) ceiling (15 min); NO: 25 ppm ($30\ mg/m^3$) TWA	Respiratory and blood effects	Pulmonary function testing required
Nitroglycerin and ethylene glycol dinitrate (EGDN) (June 1978)	Nitroglycerin: 2 mg/m^3 (0.2 ppm) ceiling (Skin); EGDN: 1 mg/m^3 (0.2 ppm) ceiling (Skin)	0.1 mg/m^3 ceiling (20 min) recommended limit for either substance alone or mixtures	Circulatory system effects	Prevent skin contact
2-Nitro-naphthalene (CIB December 1976)	None	Ca Reduce exposure to lowest feasible level	Bladder cancer	Compound metabolizes to beta-naphthylamine, a known carcinogen
2-Nitropropane (CIB April 1977; revised October 1980 in Joint OSHA/NIOSH Health Hazard Alert)	25 ppm ($90\ mg/m^3$), 8-hr TWA	Ca Reduce exposure to lowest feasible level	Potential for cancer in humans; produced liver tumors in rats	Medical monitoring with specific emphasis on liver function tests
N-Nitroso-dimethylamine (NIOSH testimony at OSHA hearing September 1973)	No PEL Cancer-suspect agent Stringent workplace controls, recordkeeping, and medical surveillance required 29 CFR 1910.1016	Ca Use 29 CFR 1910.1016	Potential for cancer in humans; produced tumors of the liver, kidney, lung, and nasal cavity in animals	None

---

*Date recommendation was published or testimony presented is in parentheses.

†NIOSH TWA recommendations are based on exposures up to 10 hours unless otherwise noted.

§Unless otherwise noted health effects cited are for humans.

		NIOSH		
Potential Hazard[*]	OSHA PEL'S/ Standard	REL'S[†]/Other Recommendations	Health Effect(s) Considered[§]	Comments
Noise (August 1972)	90 dBA, 8-hr TWA	85 dBA TWA; 115 dBA ceiling	Hearing damage	None
Organotin compounds (November 1976)	0.1 mg/m^3, 8-hr TWA	0.1 mg Sn/m^3 TWA	Eye, skin, liver, nervous system, and cardiovascular effects	Chest x ray, blood and urine monitoring, eye tests, heart examination, and nervous system testing required; prevent skin and eye contact
Paint and allied coating products, manufacture of (September 1984)	Many aspects covered under the numerous OSHA regulations for general industry (29 CFR 1910)	Various recommendations for the handling of raw materials and finished products; dispersion of pigment or resin particles; thinning, tinting, and shading; filling; and laboratory functions	Injury and a wide range of toxicities considered	Paint and allied coating products include paints, varnishes, lacquers, stains, putties, and paint and varnish removers
Parathion (June 1976)	0.1 mg/m^3, 8-hr TWA (Skin)	0.05 mg/m^3 TWA	Nervous system effects	Prevent skin contact; blood monitoring required
Pentachloroethane (CIB August 1978)	None	To be handled in the workplace with caution	Central nervous system effects; possible liver and kidney effects	Exposures should be minimized due to the structural similiarity to the carcinogenic chloroethanes
Pesticides, manufacture and formulation of (July 1978)	Current OSHA PEL's or previous NIOSH REL's to be followed; stringent work-practice and medical surveillance requirements to be instituted. Pesticides considered in groups based on toxicity.		Wide range of toxicities considered; cancer; nervous and reproductive system effects	Blood monitoring required for some groups; workers to be warned of reproductive effects for some compounds; prevent skin contact

Phenol (July 1976)	5 ppm (19 mg/m^3), 8-hr TWA (Skin)	5.2 ppm (20 mg/m^3) TWA; 15.6 ppm (60 mg/m^3) ceiling (15 min)	Skin, eye, central nervous system, liver, and kidney effects	Prevent skin and eye contact
Phenyl-beta-naphthylamine (CIB December 1976)	None	Ca Reduce exposure to lowest feasible level	Bladder cancer	Compound metabolizes to beta-naphthylamine, a known carcinogen
Phosgene (February 1976)	0.1 ppm (0.4 mg/m^3), 8-hr TWA	0.1 ppm (0.4 mg/m^3) TWA; 0.2 ppm (0.8 mg/m^3) ceiling (15 min)	Respiratory effects	Pulmonary function testing and chest x ray required
Polychlorinated biphenyls (September 1977)	42% chlorine: 1 mg/m^3, 8-hr TWA; 54% chlorine: 0.5 mg/m^3, 8-hr TWA	Ca 1 μg/m^3 TWA (the minimum reliably detectable concentration using the recommended sampling and analytical methods)	Potential for cancer in humans; produced tumors of the liver, pituitary gland and leukemias in animals; skin, liver, and reproductive system effects	Blood testing required; female workers of child-bearing age and nursing mothers to be warned of potential adverse effects
Polychlorinated biphenyls (PCB's), potential health hazards from electrical equipment fires or failures (CIB February 1986)	42% chlorine: 1 mg/m^3, 8-hr TWA; 54% chlorine: 0.5 mg/m^3, 8-hr TWA	Ca Reduce exposure to lowest feasible limit	Potential for cancer in humans; produced tumors of the liver, pituitary gland and leukemias in animals; skin, liver, and reproductive system effects	Fire-related incidents involving PCB's have resulted in widespread contamination of buildings with PCB's and, in some cases, with PCDF's and PCDD's including TCDD. Emergency response personnel, maintenance or cleanup workers, or building occupants may be exposed to these compounds.

---

[*] Date recommendation was published or testimony presented is in parentheses.

[†] NIOSH TWA recommendations are based on exposures up to 10 hours unless otherwise noted.

[§] Unless otherwise noted health effects cited are for humans.

		NIOSH		
Potential Hazard*	OSHA PEL'S/ Standard	REL'S†/Other Recommendations	Health Effect(s) Considered§	Comments
Precast concrete products industry, comprehensive safety recommendations for (Technical Guideline June 1984)	Many aspects covered under numerous OSHA regulations for general industry (29 CFR 1910)	Various recommendations for safe work practices and worker training	Injury and death	Equipment, conditions, and many of the tasks specific to the industry are not covered under the existing regulations
beta-Propiolactone (NIOSH testimony at OSHA hearing September 1973)	No PEL Cancer-suspect agent Stringent workplace controls, recordkeeping, and medical surveillance required 29 CFR 1910.1013	Ca Use 29 CFR 1910.1013	Potential for cancer in humans; produced tumors of the liver, skin, and stomach in animals	None
Refined petroleum solvents (July 1977)	2,900 mg/m^3 (500 ppm), 8-hr TWA (Stoddard solvent)	Kerosene: 100 mg/m^3 TWA; all other solvents: 350 mg/m^3 TWA; 1,800 mg/m^3 ceiling (15 min)	Eye, nose, and throat irritation; dermatitis; nervous system effects	Blood and urine monitoring required; action level for petroleum ether, rubber solvent, naphtha: 200 mg/m^3 TWA; action level for mineral spirits and Stoddard solvent: 350 mg/m^3 TWA; action level for kerosene: 100 mg/m^3 TWA; prevent skin contact
Silica, crystalline (November 1974)	250/%SiO$_2$+5 in mppcf, or 10 mg/m^3/%SiO$_2$+2 (respirable quartz)	50 μg/m^3 TWA, respirable free silica	Chronic lung disease (silicosis)	Chest x ray, pulmonary function testing required
Sodium hydroxide (September 1975)	2 mg/m^3, 8-hr TWA	2 mg/m^3 ceiling (15 min)	Respiratory irritation	Prevent skin and eye contact

Styrene (September 1983)	100 ppm, 8-hr TWA; 200 ppm acceptable ceiling; 600 ppm maximum ceiling (5 min in 3 hr)	50 ppm (213 mg/m^3) TWA; 100 ppm (426 mg/m^3) ceiling	Nervous system effects; eye and respiratory system irritation; reproductive system effects	Prevent skin contact; workers to be warned of possible adverse reproductive effects
Sulfur dioxide (February 1974; revised May 1977 as part of NIOSH testimony at OSHA hearing)	5 ppm (13 mg/m^3), 8-hr TWA	0.5 ppm (1.3 mg/m^3) TWA	Respiratory effects	Pulmonary function testing required
Sulfuric acid (June 1974)	1 mg/m^3, 8-hr TWA	1 mg/m^3 TWA	Pulmonary irritation	Prevent skin and eye contact
2,3,7,8-Tetra-chlorodibenzo-*p*-dioxin (TCDD) (CIB January 1984)	None	Ca Reduce exposure to lowest feasible level	Potential for cancer in humans; produced tumors at many sites in animals; chloracne	None
1,1,1,2-Tetra-chloroethane (CIB August 1978)	None	To be handled in the workplace with caution	Central nervous system effects; possible liver and kidney effects	Exposures should be minimized due to the structural similiarity to the carcinogenic chloroethanes
1,1,2,2-Tetra-chloroethane (December 1976; revised in CIB August 1978)	5 ppm (35 mg/m^3), 8-hr TWA (Skin)	Ca Reduce exposure to lowest feasible level	Potential for cancer in humans; produced tumors of the liver in animals; liver, gastrointestinal, and nervous system effects	Prevent skin contact; blood monitoring required

---

[*] Date recommendation was published or testimony presented is in parentheses.

[†] NIOSH TWA recommendations are based on exposures up to 10 hours unless otherwise noted.

[§] Unless otherwise noted health effects cited are for humans.

		NIOSH		
Potential Hazard*	OSHA PEL'S/ Standard	REL'S†/Other Recommendations	Health Effect(s) Considered§	Comments
Tetrachloroethylene (July 1976; revised January 1978 in CIB)	100 ppm, 8-hr TWA; 200 ppm acceptable ceiling; 300 ppm maximum ceiling (5 min in 3 hr)	Ca Minimize workplace exposure levels; limit number of workers exposed	Potential for cancer in humans; produced tumors of the liver in animals	None
Thiols: n-alkane mono thiols, cyclohexanethiol, and benzenethiol (September 1978)	Butylmercaptan: (1-butanethiol) 10 ppm (35 mg/m^3), 8-hr TWA; ethylmercaptan (1-ethanethiol): 10 ppm (25 mg/m^3) ceiling; methylmercaptan (1-methanethiol): 10 ppm (20 mg/m^3) ceiling	All values in ppm (mg/m^3) ceilings (15 min): 1-methanethiol: 0.5 (1.0); 1-ethanethiol: 0.5 (1.3); 1-propanethiol: 0.5 (1.6); 1-butanethiol: 0.5 (1.8); 1-pentanethiol: 0.5 (2.1); 1-hexanethiol: 0.5 (2.4); 1-heptanethiol: 0.5 (2.7); 1-octanethiol: 0.5 (3.0); 1-nonanethiol: 0.5 (3.3); 1-decanethiol: 0.5 (3.6); 1-undecanethiol: 0.5 (3.9); 1-dodecanethiol: 0.5 (4.1); 1-hexadecanethiol: 0.5 (5.3); 1-octadecanethiol: 0.5 (5.9); cyclohexanethiol: 0.5 (2.4), benzenethiol: 0.1 (0.5); Mixtures of thiols to be controlled by calculation of equivalent concentrations	Irritation; eye, skin, blood, and nervous system effects	Blood and urine monitoring required; prevent skin contact

o-Tolidine (August 1978)	None	Ca 20 $\mu g/m^3$ ceiling (60 min)	Bladder cancer; nasal irritation	Urine testing required; quarterly urine monitoring recommended; prevent skin contact
o-Tolidine-based dyes (Joint NIOSH/OSHA Health Hazard Alert December 1980)	None	Ca Should be handled in the workplace with caution; minimize exposures	Bladder cancer	Substitute less toxic dyes wherever possible
Toluene (January 1974)	200 ppm, 8-hr TWA; 300 ppm acceptable ceiling; 500 ppm maximum ceiling (10 min)	100 ppm (375 mg/m^3), 8-hr TWA; 200 ppm (750 mg/m^3) ceiling (10 min)	Central nervous system depressant	None
Toluene diisocyanate (July 1973; revised—see Diisocyanates September 1978)	0.02 ppm (0.14 mg/m^3) ceiling	0.005 ppm (0.036 mg/m^3) TWA; 0.02 ppm (0.14 mg/m^3) ceiling (20 min)	Respiratory effects	Chest x ray, blood tests, pulmonary function testing required
1,1,1-Tri-chloroethane (July 1976)	350 ppm (1,900 mg/m^3), 8-hr TWA	350 ppm (1,910 mg/m^3) ceiling (15 min); action level set at 200 ppm (1,091 mg/m^3) TWA	Central nervous system, liver, and cardiovascular effects	Medical warning of possible congenital abnormalities required
1,1,2-Tri-chloroethane (CIB August 1978)	10 ppm (45 mg/m^3), 8-hr TWA (Skin)	Ca Reduce exposure to lowest feasible level	Potential for cancer in humans; produced liver tumors in animals; central nervous system effects	None

---

[*] Date recommendation was published or testimony presented is in parentheses.

[†] NIOSH TWA recommendations are based on exposures up to 10 hours unless otherwise noted.

[§] Unless otherwise noted health effects cited are for humans.

Potential Hazard*	OSHA PEL'S/ Standard	NIOSH REL'S†/Other Recommendations	NIOSH Health Effect(s) Considered§	NIOSH Comments
Trichloroethylene (July 1973; revised in Special Hazard Review January 1978)	100 ppm, 8-hr TWA; 200 ppm acceptable ceiling; 300 ppm maximum ceiling (5 min in 2 hr)	Ca 25 ppm TWA	Potential for cancer in humans; produced liver tumors in animals; central nervous system effects	Workers to be warned of hazards; 25 ppm level can be achieved by use of existing engineering control technology
Trimellitic anhydride (CIB February 1978)	None	Should be handled in the workplace as an extremely toxic substance	Pulmonary edema; immunologic sensitization; irritation of pulmonary tract, eyes, nose, and skin	Limit exposure to as few workers as possible while minimizing workplace levels
Tungsten and cemented tungsten carbide (September 1977)	None	Insoluble tungsten: 5 mg W/m^3 TWA; soluble tungsten: 1 mg W/m^3 TWA; dust of cemented tungsten carbide (containing > 2% cobalt): 0.1 mg Co/m^3 TWA; dust of cemented tungsten carbide (containing > 0.3% nickel): 15 μg Ni/m^3 TWA	Lung and skin effects	Pulmonary function testing and chest x ray required
Ultraviolet radiation (December 1972)	None	For spectral region of 315-400 nm: 1.0 mW/cm^2 for periods >1,000 sec; for periods $\leq$1,000 sec, 1,000 mW · sec/cm^2 (1.0 J/cm^2); for spectral region 200-315 nm: consult criteria document	Skin and eye effects	None
Vanadium (August 1977)	Vanadium pentoxide (dust): 0.5 mg/m^3 ceiling; (fume): 0.1 mg/m^3 ceiling; ferrovanadium: 1 mg/m^3, 8-hr TWA	Vanadium compounds: 0.05 mg V/m^3 ceiling (15 min); metallic vanadium and vanadium carbide: 1 mg V/m^3 TWA	Eye, skin, and lung effects	Pulmonary function testing and chest x ray required

Vibration syndrome (CIB March 1983)	None	Jobs should be redesigned to minimize the use of vibrating handtools; powered handtools should be redesigned to minimize vibration	Vibration syndrome; adverse circulatory and neural effects in the fingers	None
Vinyl acetate (September 1978)	None	4 ppm (15 mg/m^3) ceiling (15 min)	Irritation	None
Vinyl chloride (March 1974; reaffirmed June 1974 as part of NIOSH testimony at OSHA hearing)	1 ppm, 8-hr TWA; 5 ppm ceiling (15 min) 29 CFR 1910.1017	Ca Lowest reliably detectable level	Liver cancer	Liver function testing required
Vinyl halides (September 1978)	None except for vinyl chloride	Ca Vinyl halides to be controlled as specified for vinyl chloride in 29 CFR 1910.1017 with eventual goal of zero exposure	Liver cancer for vinyl chloride; potential for cancer from the other vinyl halides that have produced liver and kidney tumors in animals	Vinyl halides include vinyl chloride, vinylidene chloride, vinyl bromide, vinyl fluoride, and vinylidene fluoride monomers
Waste anesthetic gases and vapors (March 1977)	None for substances when used as anesthetic agents	Halogenated anesthetic agents: 2 ppm ceiling (1 hr); nitrous oxide: 25 ppm TWA during periods of use	Reproductive system effects and audiovisual performance decrements	Workers to be advised of potential effects; abnormal outcome of pregnancies of workers and spouses to be documented
Xylene (May 1975)	100 ppm (435 mg/m^3), 8-hr TWA	100 ppm (434 mg/m^3) TWA; 200 ppm (868 mg/m^3) ceiling (10 min)	Central nervous system depressant; respiratory irritation	None
Zinc oxide (October 1975)	5 mg ZnO/m^3, 8-hr TWA	5 mg ZnO/m^3 TWA; 15 mg ZnO/m^3 ceiling (15 min)	Metal fume fever	None

---

[*] Date recommendation was published or testimony presented is in parentheses.

[†] NIOSH TWA recommendations are based on exposures up to 10 hours unless otherwise noted.

[§] Unless otherwise noted health effects cited are for humans.

Director, Centers for Disease Control
James O. Mason, M.D., Dr.P.H.
Director, Epidemiology Program Office
Carl W. Tyler, Jr., M.D.

Editor
Michael B. Gregg, M.D.

☆U.S. Government Printing Office: 1986-746-149/40025 Region IV

UNITED STATES GOVERNMENT PRINTING OFFICE
SUPERINTENDENT OF DOCUMENTS
Washington, D.C. 20402

OFFICIAL BUSINESS
Penalty for Private Use, $300

BULK RATE
POSTAGE & FEES PAID
GPO
Permit No. G-26

MMWR NEWAROOON DR 376 F 1 *
NEWARK PUBLIC LIBR *
5 WASHINGTON ST *
NEWARK NJ 07101 *

HHS Publication No. (CDC) 86-8017

Redistribution using indicia is illegal.

35/1SS

CENTERS FOR DISEASE CONTROL

MMWR

MORBIDITY AND MORTALITY WEEKLY REPORT

Vol. 35/No. 1SS

CDC Surveillance Summaries 1986

Contents

Hysterectomy among Women of Reproductive Age, United States, Update for 1981-1982

Foodborne Disease Outbreaks, Annual Summary, 1982

Nosocomial Infection Surveillance, 1984

NEWARK PUBLIC LIBRARY

JUL 9 1986

U. S. GOVT. PUBS.
DEPOSITORY 378

U.S. DEPARTMENT OF HEALTH AND HUMAN SERVICES
PUBLIC HEALTH SERVICE
CENTERS FOR DISEASE CONTROL
ATLANTA, GEORGIA 30333

HE 20.7009:

CDC Surveillance Summaries are published by the Epidemiology Program Office, Centers for Disease Control, Public Health Service, U.S. Department of Health and Human Services, Atlanta, Georgia 30333.

SUGGESTED CITATIONS

General: Centers for Disease Control. *CDC Surveillance Summaries*. *MMWR* 1986;35(No. 1SS).

Specific: Centers for Disease Control. [Title of particular article/chapter.] In: *CDC Surveillance Summaries*. *MMWR* 1986;35(No. 1SS):[inclusive page numbers].

Centers for Disease Control .... James O. Mason, M.D., Dr.P.H.,
Director

This report was prepared by:

Epidemiology Program Office .... Carl W. Tyler, Jr., M.D.,
Director

Michael B. Gregg, M.D.,
Deputy Director for Communications

Editorial Services .... R. Elliott Churchill, M.A.,
Chief
Consulting Editor

Patsy H. Hurst,
Illustrator

Lynne McIntyre,
Coordinator

Division of Surveillance and
Epidemiologic Studies .... Stephen B. Thacker, M.D.,
Director

Thomas P. Whitley, Jr.,
Computer Graphics Specialist

Table of Contents

Foreword .... iiSS

History of Centers for Disease Control Surveillance Activities .... iiiSS

Data Sources .... ivSS

Current Surveillance Publications .... vSS

Contributors to *CDC Surveillance Summaries*

Hysterectomy among Women of Reproductive Age, United States, Update for 1981-1982
Kathleen L. Irwin, M.D., Joyce M. Hughes, Sara W. Gill 1SS

Foodborne Disease Outbreaks, Annual Summary, 1982
Kristine L. MacDonald, M.D., Patricia M. Griffin, M.D. 7SS

Nosocomial Infection Surveillance, 1984
Teresa C. Horan, M.P.H., John W. White, Ph.D., William R. Jarvis, M.D., T. Grace Emori, R.N., M.S., David H. Culver, Ph.D., Van P. Munn, B.S. Clyde Thornsberry, Ph.D., David R. Olson, Ph.D., James M. Hughes, M.D 17SS

State and Territorial Epidemiologists and State Laboratory Directors .... Inside Back Cover

Foreword

The purpose of the *CDC Surveillance Summaries* is to make available the most current information on conditions of public health interest for which CDC has major responsibility. The *CDC Surveillance Summaries* provide detailed analysis of the most current available data obtained for CDC surveillance programs. These reports complement other data published by CDC in the *Morbidity and Mortality Weekly Report (MMWR)*, the *MMWR Annual Summary*, and various disease-surveillance reports. This volume contains epidemiologic information derived from surveillance forms, special investigations, and other sources of information collected at the state and national levels.

History of CDC Surveillance Activities

CDC has been actively involved in disease-surveillance activities since the formation of the Communicable Disease Center in 1946. The original scope of the National Surveillance Program included the study of malaria, murine typhus, smallpox, psittacosis, diphtheria, leprosy, and sylvatic plague. In 1954, a surveillance section was established within the Epidemiology Branch of CDC, primarily concerned with planning and conducting continuing surveillance and making periodic reports. National emergencies such as the Asian influenza pandemic and the discovery of Legionnaires' disease have prompted the involvement of CDC in new surveillance activities. Over the years the surveillance activities of CDC have expanded to include not only new areas in infectious disease but also programs in human reproduction, environmental health, chronic disease, risk reduction, and occupational safety and health. Ongoing evaluation of these programs has led to new methods of data collection and analysis and has prompted examination of how data are disseminated to the public health community.

In 1980 and 1981, a survey of CDC staff and state epidemiologists suggested that improved coordination of surveillance reports with the *MMWR* and the *MMWR Annual Summary* would facilitate timely publication; provide greater uniformity in the acquisition, evaluation, and reporting of surveillance data; and encourage use of these data. Several approaches to the development of a systematic process of disseminating disease-specific surveillance reports were considered. On the basis of considerations of timeliness, cost advantages, and editorial uniformity, a report published on a quarterly basis was recommended. Subsequent financial and personnel constraints have made it necessary to publish these reports less frequently.

The *CDC Surveillance Summaries* contain information more reflective of the detailed surveillance reports of the past. CDC hopes that the *Surveillance Summaries* will disseminate surveillance data on a regular schedule, improve the clarity of community public health information, and also produce a cost savings. Although the *CDC Surveillance Summaries* are published more often than once a year, they will typically contain annual data rather than interim data. The *MMWR Annual Summary* will complement rather than serve as the cumulative summary of the *Surveillance Summaries*.

Data Sources

Data on the reported occurrence of notifiable diseases are derived from reports supplied by the state and territorial departments of health and CDC program activities, routinely published in the *MMWR*, and compiled in final form in the *MMWR Annual Summary*.

CDC also maintains national surveillance programs for selected diseases with the cooperation of state and local health departments as well as other federal agencies, and publishes detailed epidemiologic analyses periodically. Data appearing in the *CDC Surveillance Summaries* or in a surveillance report may not agree exactly with reports published in the *MMWR* because of differences in timing of reports or because of refinements in case definition. It should be noted that data collected for the *MMWR* and the more detailed data published by individual CDC programs are collected independently.

These data should be interpreted with caution. Some diseases that cause severe clinical illness and are associated with serious consequences are probably reported quite accurately. However, diseases that are clinically mild and infrequently associated with serious consequences are less likely to be reported. Additionally, subclinical cases are seldom detected except in the course of epidemic investigations or special studies. The degree of completeness of reporting is also influenced by the diagnostic facilities available, the control measures in effect, and the interests and priorities of state and local officials responsible for disease control and surveillance. Finally, factors such as the introduction of new diagnostic tests and the discovery of new disease entities may cause changes in disease reporting independent of the true incidence of disease. Despite these limitations the data in these reports have proven to be useful in the analysis of trends.

Surveillance Programs
Centers for Disease Control

Surveillance program	Responsible branch	Most recent report/summary*
Abortion	Pregnancy Epidemiology Branch Division of Reproductive Health Center for Health Promotion and Education	November 1985 (1981 data)
Behavioral risk factors	Division of Nutrition Center for Health Promotion and Education	1984 (SS 33/1) (data from 1981-1983)
Berylliosis cohorts: registry of disease and exposure	Surveillance Branch Division of Surveillance, Hazard Evaluations, and Field Studies National Inst. for Occup. Safety & Hlth.	March 1983 (data from 1951-1980)
Biologics	Data Management Branch Division of Immunization Center for Prevention Services	December 1982 (1982 data)
Botulism	Enteric Diseases Branch Division of Bacterial Diseases Center for Infectious Diseases	May 1979 (data from 1899-1977)
Brucellosis	Bacterial Zoonoses Activity Division of Bacterial Diseases Center for Infectious Diseases	June 1979 (1978 data)
Coal workers' pneumoconiosis	Epidemiological Investigations Branch Division of Respiratory Disease Studies National Inst. for Occup. Safety & Hlth.	1985 (SS 34/1) (data from 1970-1981)
Congenital malformations	Birth Defects Branch Chronic Diseases Division Center for Environmental Health	1985 (SS 34/2) (data from 1970-1983)
Dengue	Dengue Branch Division of Vector-Borne Viral Diseases Center for Infectious Diseases	1985 (SS 34/2) (data from 1983-1984)
Diabetes	Division of Diabetes Control Center for Prevention Services	June 1979 (1978 data)
Diphtheria	Surveillance, Investigations and Research Branch Division of Immunization Center for Prevention Services	July 1978 (data from 1971-1975)
Ectopic pregnancy	Pregnancy Epidemiology Branch Division of Reproductive Health Center for Health Promotion and Education	1984 (SS 33/2) (data from 1979-1980)

---

*Publications denoted by "SS" appeared in issues of *CDC Surveillance Summaries*. Other reports listed can be obtained by contacting the responsible administrative unit listed.

Surveillance Programs
Centers for Disease Control

Surveillance program	Responsible branch	Most recent report/summary*
Encephalitis	Arbovirus Reference Branch Division of Vector-Borne Viral Diseases Center for Infectious Diseases	May 1981 (1978 data)
Enterovirus	Respiratory and Enterovirus Branch Division of Viral Diseases Center for Infectious Diseases	November 1981 (data from 1970-1979)
Fifteen leading causes of death in the U.S., 1978	Health Analysis and Planning for Preventive Services Center for Prevention Services	September 1982 (1978 data)
Foodborne disease	Enteric Diseases Branch Division of Bacterial Diseases Center for Infectious Diseases	1985 (SS 34/3) (1982 data)
Gonorrhea	Division of Sexually Transmitted Diseases Center for Prevention Services	1984 (SS 33/4) (data from 1983-1984)
Hepatitis	Hepatitis Branch Division of Viral Diseases Center for Infectious Diseases	1985 (SS 34/1) (data from 1982-1983)
Homicide	Violence Epidemiology Branch Office of the Director Center for Health Promotion and Education	May 1983 (SS 32/2) (data from 1970-1978)
Hysterectomy	Epidemiologic Studies Branch Division of Reproductive Health Center for Health Promotion and Education	1985 (SS 34/3) (data from 1981-1982)
Influenza	Influenza Branch Division of Viral Diseases Center for Infectious Diseases	July 1984 (data from 1983-1984)
Lead poisoning in workers	Surveillance Branch Division of Surveillance, Hazard Evaluations, and Field Studies National Inst. for Occup. Safety & Hlth.	April 1983 (data from 1976-1980)
Leprosy	Respiratory and Special Pathogens Branch Division of Bacterial Diseases Center for Infectious Diseases	April 1976 (data from 1971-1973)
Leptospirosis	Bacterial Zoonoses Activity Division of Bacterial Diseases Center for Infectious Diseases	August 1979 (1978 data)

---

*Publications denoted by "SS" appeared in issues of *CDC Surveillance Summaries*. Other reports listed can be obtained by contacting the responsible administrative unit listed.

Surveillance Programs
Centers for Disease Control

Surveillance program	Responsible branch	Most recent report/summary*
Malaria	Malaria Branch Division of Parasitic Diseases Center for Infectious Diseases	October 1984 (1983 data)
Maternal mortality	Division of Reproductive Health Center for Health Promotion and Education	1984 (SS 33/1) (data from 1974-1978)
Measles	Surveillance, Investigations and Research Branch Division of Immunization Center for Prevention Services	September 1982 (data from 1977-1981)
Mumps	Surveillance, Investigations and Research Branch Division of Immunization Center for Prevention Services	July 1978 (data from 1974-1976)
National electronic injury surveillance system	Safety Surveillance Branch Division of Safety Research National Inst. for Occup. Safety & Hlth.	May 1983 (SS 32/2) (1982 data)
National Occupational Hazard Survey (NOHS)	Surveillance Branch Division of Surveillance, Hazard Evaluations, and Field Studies National Inst. for Occup. Safety & Hlth.	NIOSH Technical Report DHHS (NIOSH) Pub. No. 83-117
Nosocomial infections	National Nosocomial Infections Surveillance System Hospital Infections Program Center for Infectious Diseases	1985 (SS 34/3) (1984 data)
Nutrition	Division of Nutrition Center for Health Promotion and Education	November 1982 (1980 data)
Occupational characteristics of disabled workers	Surveillance Branch Division of Surveillance, Hazard Evaluations, and Field Studies National Inst. for Occup. Safety & Hlth.	July 1980 (data from 1969-1978)
Occupational hazard surveillance	Surveillance Branch Division of Surveillance, Hazard Evaluations, and Field Studies National Inst. for Occup. Safety & Hlth.	1985 (SS 34/2) (data from 1970-1982)
Occupational injuries among loggers	Safety Surveillance Branch Division of Safety Research National Inst. for Occup. Safety & Hlth.	August 1983 (SS 32/3) (data from 1969-1974)

---

*Publications denoted by "SS" appeared in issues of *CDC Surveillance Summaries*. Other reports listed can be obtained by contacting the responsible administrative unit listed.

Surveillance Programs
Centers for Disease Control

Surveillance program	Responsible branch	Most recent report/summary*
Occupational injuries in the meatpacking industry	Safety Surveillance Branch Division of Safety Research National Inst. for Occup. Safety & Hlth.	1985 (SS 34/1) (data from 1976-1981)
Occupational mortality in Washington State	Surveillance Branch Division of Surveillance, Hazard Evaluations, and Field Studies National Inst. for Occup. Safety & Hlth.	DHHS (NIOSH) Pub. No. 83-116 (data from 1950-1979)
Pediatric nutrition	Division of Nutrition Center for Health Promotion and Education	1983 (SS 32/4) (1982 data)
Pelvic inflammatory disease	Division of Sexually Transmitted Disease Center for Prevention Services	1983 (SS 32/4) (data from 1965-1982)
Plague	Plague Branch Division of Vector-Borne Viral Diseases Center for Infectious Diseases	1985 (SS 34/2) (1984 data)
Poliomyelitis	Surveillance, Investigations and Research Branch Division of Immunization Center for Prevention Services	December 1982 (data from 1980-1981)
Psittacosis	Bacterial Zoonoses Activity Division of Bacterial Diseases Center for Infectious Diseases	February 1983 (SS 32/1) (1979 data)
Rabies	Viral and Rickettsial Zoonoses Branch Division of Viral Diseases Center for Infectious Diseases	1985 (SS 34/1) (1983 data)
Reye syndrome	Epidemiology Office Division of Viral Diseases Center for Infectious Diseases	1984 (SS 33/3) (1983 data)
Rickettsial disease (RMSF, murine typhus, Q fever, endemic typhus)	Viral and Rickettsial Zoonoses Branch Division of Viral Diseases Center for Infectious Diseases	May 1981 (1979 data)
Rocky mountain spotted fever Division of Viral Diseases	Viral and Rickettsial Zoonoses Branch Center for Infectious Diseases	1984 (SS 33/3) (data from 1981-1983)
Rubella	Surveillance, Investigations and Research Branch Division of Immunization Center for Prevention Services	1984 (SS 33/4) (1983 data)

---

*Publications denoted by "SS" appeared in issues of *CDC Surveillance Summaries*. Other reports listed can be obtained by contacting the responsible administrative unit listed.

Surveillance Programs
Centers for Disease Control

Surveillance program	Responsible branch	Most recent report/summary*
Salmonella	Enteric Diseases Branch Division of Bacterial Diseases Center for Infectious Diseases	December 1982 (1980 data)
Sentinel health event (occupational) (SHE)	Surveillance Branch Division of Surveillance, Hazard Evaluations, and Field Studies National Inst. for Occup. Safety & Hlth.	September 1983
Summer mortality	Special Studies Branch Chronic Diseases Division Center for Environmental Health	February 1983 (SS 32/1) (data from 1979-1981)
Surgical sterilization	Epidemiologic Studies Branch Division of Reproductive Health Center for Health Promotion and Education	August 1983 (SS 32/3) (data from 1979-1980)
Toxic-shock syndrome	Respiratory and Special Pathogens Branch Division of Bacterial Diseases Center for Infectious Diseases	1984 (SS 33/3) (data from 1960-1984)
Trichinosis	Helminthic Diseases Branch Division of Parasitic Diseases Center for Infectious Diseases	1984 (SS 33/4) (1983 data)
Tuberculosis	Division of Tuberculosis Control Center for Prevention Services	July 1985 (1984 data) TB Statistics: States & Cities
		November 1983 (1980 data) TB in the United States
U.S. immunization survey	Surveillance, Investigations and Research Branch Division of Immunization Center for Prevention Services	April 1983 (data from 1979-1982)
Venereal disease	Division of Sexually Transmitted Disease Center for Prevention Services	(1980 data) Sexually Transmitted Diseases Statistical Letter-No. 130
		(data from 1978-1979) STD Fact Sheet-Edition 35
Water-related disease outbreaks	Enteric Diseases Branch Division of Bacterial Diseases Center for Infectious Diseases	September 1984 (1983 data)

---

*Publications denoted by "SS" appeared in issues of *CDC Surveillance Summaries*. Other reports listed can be obtained by contacting the responsible administrative unit listed.

Contributors to *CDC Surveillance Summaries*

Center for Health Promotion and Education, Dennis D. Tolsma, M.P.H., *Director*
  Division of Reproductive Health, James S. Marks, M.D., *Acting Director*
    Epidemiologic Studies Branch, George L. Rubin, M.B., F.R.A.C.P., *Chief*
    Research and Statistics Branch, Jack C. Smith, M.S., *Chief*
    Educational Resources Branch, Priscilla B. Holman, M.S. Ed., Chief
Center for Infectious Diseases, Walter R. Dowdle, Ph.D., *Director*
  Division of Bacterial Diseases, John C. Feeley, Ph.D., *Director*
    Enteric Diseases Branch, Paul A. Blake M.D., *Chief*
  Hospital Infections Program, James M. Hughes, M.D., *Director*
  Publications and Graphics, Frances H. Porcher, M.A., *Chief*

Hysterectomy among Women of Reproductive Age, United States, Update for 1981-1982

Kathleen L. Irwin, M.D.
Herbert B. Peterson, M.D.
Epidemiologic Studies Branch
Joyce M. Hughes
Sara W. Gill
Research and Statistics Branch
Division of Reproductive Health
Center for Health Promotion and Education

Introduction

Nearly 5 million American women underwent hysterectomy in the last decade, making this procedure one of the most frequently performed operations for women of reproductive age (*1*) and an important public health concern. Since 1970, CDC has provided epidemiologic surveillance of hysterectomies performed on women 15-44 years of age; summary data have been previously published (*2-4*). This report provides an analysis of the influence of age, race, geographic region, and surgical approach on hysterectomy rates for 1981-1982 and updates the analysis of hysterectomy rates for 1970-1980 (*4*). This previous analysis showed that the nationwide hysterectomy rate declined from 1975 to 1980 among women of reproductive age. The current analysis, however, indicates that the noted decline may have begun to plateau, although rates in 1982 were the lowest since 1970. Major differences in rates by region and age and in the percentage of hysterectomies performed by the vaginal route have persisted. In 1982, for the first time since surveillance began in 1970, blacks had a lower hysterectomy rate than whites.

Methods

The methods of this study have been described in detail elsewhere (*2*). In brief, the data were collected by the National Center for Health Statistics (NCHS) as part of the ongoing National Hospital Discharge Survey (NHDS), which estimates the number and characteristics of patients admitted to U.S. non-Federal, short-stay hospitals for surgical procedures, disease, or injury. For this and previous reports, only total and subtotal hysterectomies with or without concomitant oophorectomy were included; radical hysterectomies and pelvic exenteration were excluded.

The population estimates used in computing rates for this report were based on data from current population surveys conducted by the U.S. Bureau of the Census. Data on race were grouped into the categories of white and black, the latter category including all races other than white. In 1981, NCHS did not report the percentage of hysterectomies classified as unknown for race. Prior to release of data, unknowns were redistributed between the categories of white and black based on the distribution of race in the geographic area of the hospital and on information on the diagnoses and expected source of payment indicated on those records for which race was reported (*5*). This was not done for the 1982 data. In 1982, as for all previous years except 1981, CDC redistributed the 9% of hysterectomies classified as unknown for race according to the percentage of knowns for race in that year (*2-4*).

Results

In the period 1970-1982, an estimated 5,170,000 women 15-44 years of age underwent hysterectomy in U.S. non-Federal, short-stay hospitals. The number of hysterectomies increased from a low of 306,000 in 1970 to a high of 442,000 in 1977 and then declined to 421,000 in 1981 and 407,000 in 1982 (Figure 1).

The hysterectomy rate in 1981 was 7.9/1,000 women ages 15-44 years and in 1982 was 7.5, the lowest rate since 1970 (Figure 2). From 1970 to 1981, the hysterectomy rate for black women was higher than that for white women. However, in 1982, for the first time since surveillance began in 1970, the rate for black women, 6.7/1,000, was less than the 7.7 rate for white women. From 1975 to 1982, the hysterectomy rate declined 14% for whites and 34% for blacks. In the period 1981-1982, the average age at the time of hysterectomy was 35 years for both blacks and whites.

For the period 1981-1982, as in previous years, the highest hysterectomy rate for women of reproductive age was for the 35- to 44-year age group, and the lowest was for the 15- to 24-year age group (Figure 3). In 1982, approximately one in 63 women 35-44 years of age underwent a hysterectomy. This is a conservative estimate because women who had had hysterectomies in previous years were not removed from the denominator.

Hysterectomy rates also varied by region during this period (Figure 4). As in previous years, women in the Northeast had the lowest hysterectomy rate of the four regions, whereas women in the South had the highest rate. The hysterectomy rate for women in the South was 2.5 times that for women in the Northeast. The average age at time of hysterectomy was similar in all geographic areas, ranging from 34.2 years in the South to 36.6 years in the Northeast.

FIGURE 1. Number of hysterectomies performed in hospitals on women ages 15-44 years, United States, 1970-1982

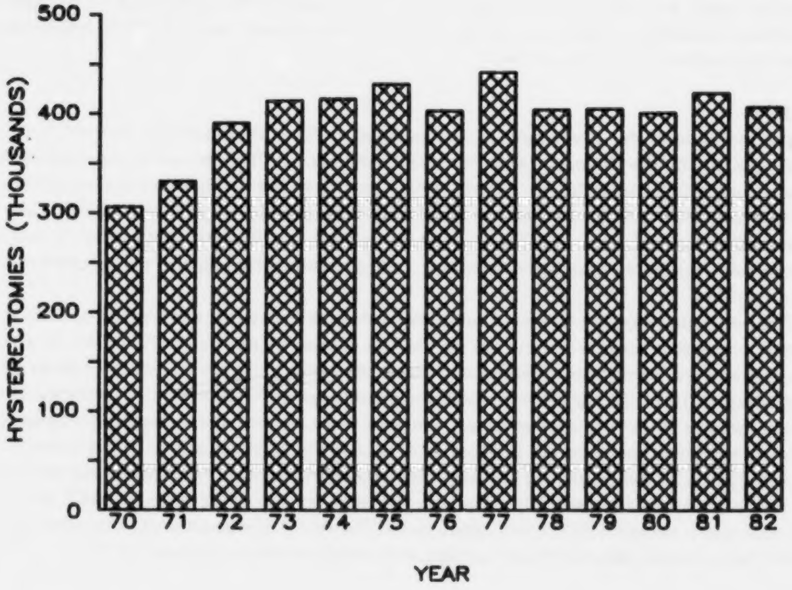


FIGURE 2. Hysterectomy rates by race, United States, 1970-1982

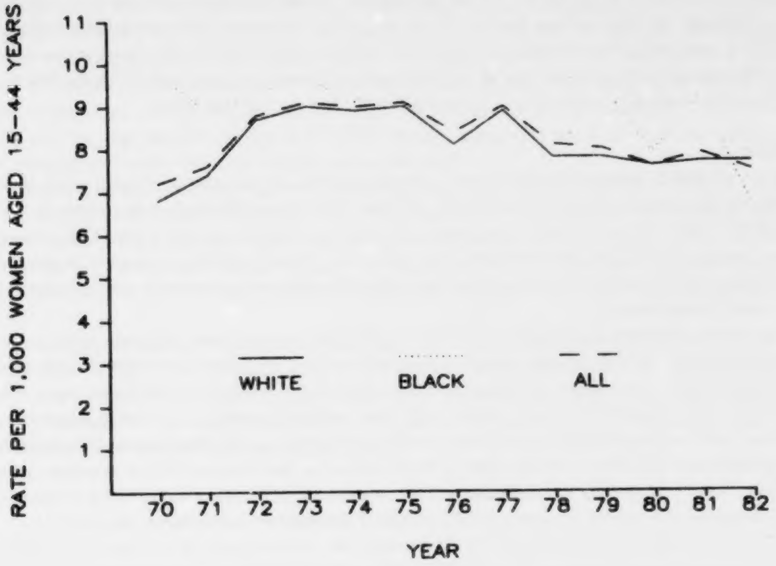
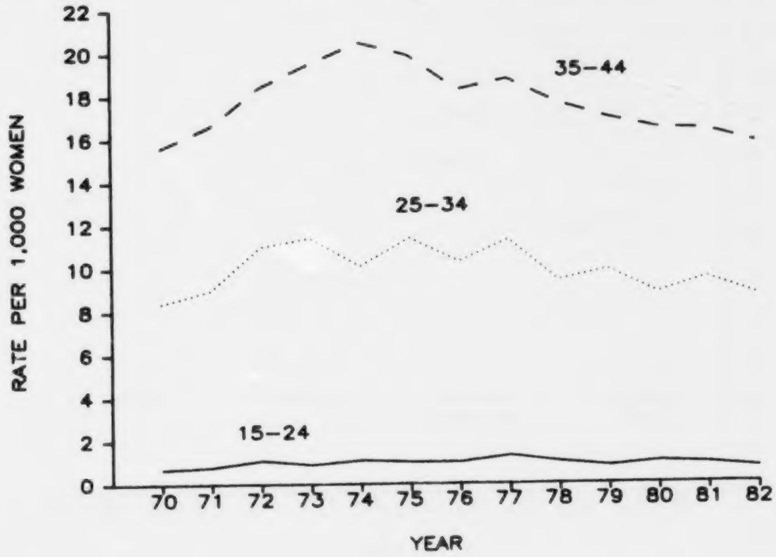


FIGURE 3. Hysterectomy rates by age group, United States, 1970-1982



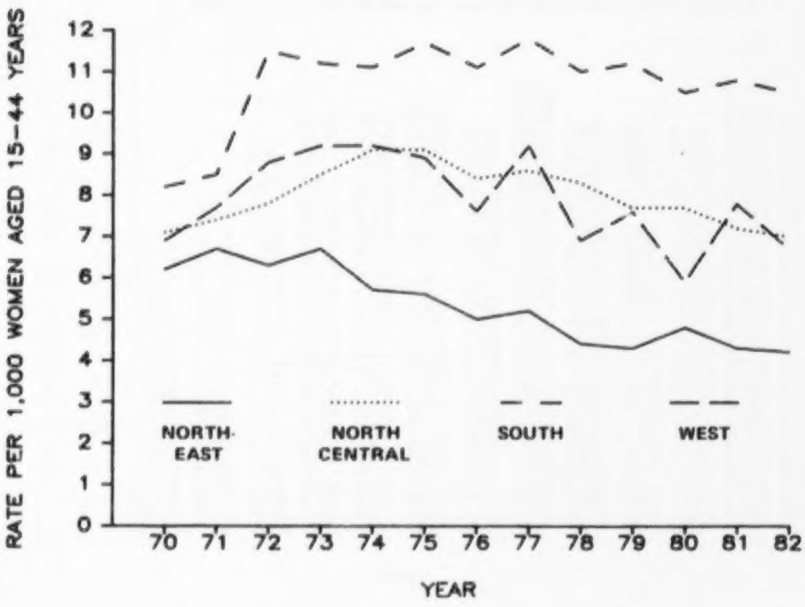
In 1981 and 1982, 27% and 24%, respectively, of all hysterectomies were performed by the vaginal route (Figure 5). In 1970, the percentage of vaginal hysterectomies among whites (27%) was similar to that among blacks (25%). By 1982, however, the percentage among whites (26%) was twice that among blacks (13%). In the period 1981-1982, there were substantial differences in the percentage of vaginal hysterectomies by region: 14% in the Northeast, 23% in the North Central region, 27% in the South, and 33% in the West.

Discussion

Analysis of NHDS data on hysterectomy among women of reproductive age shows that the decline in the nationwide rate noted from 1975 to 1980 may have begun to plateau in the period 1981-1982. However, since the rates for 1981 and 1982 represent only two annual estimates among the 13 annual estimates that have now been reported, it cannot be ascertained if these new estimates represent the beginning of a plateau in rates or a continuation of the previously noted decline.

These results probably underestimate the true rates of hysterectomy because women undergoing hysterectomy in Federally operated hospitals are not included in the NHDS data, and women ages 15-44 years who had already had a hysterectomy were not excluded from the denominator. The degree to which these rates are underestimated is probably greater in those areas and for those ages that had relatively higher rates of hysterectomy. However, a study that adjusted the 1971-1978 hysterectomy rates for the number of U.S. women ages 15-44 years who had already had a hysterectomy showed that trends in rates were similar to those reported for this period in previous CDC surgical sterilization surveillance reports (*6*).

FIGURE 4. Hysterectomy rates by geographic region, United States, 1970-1982

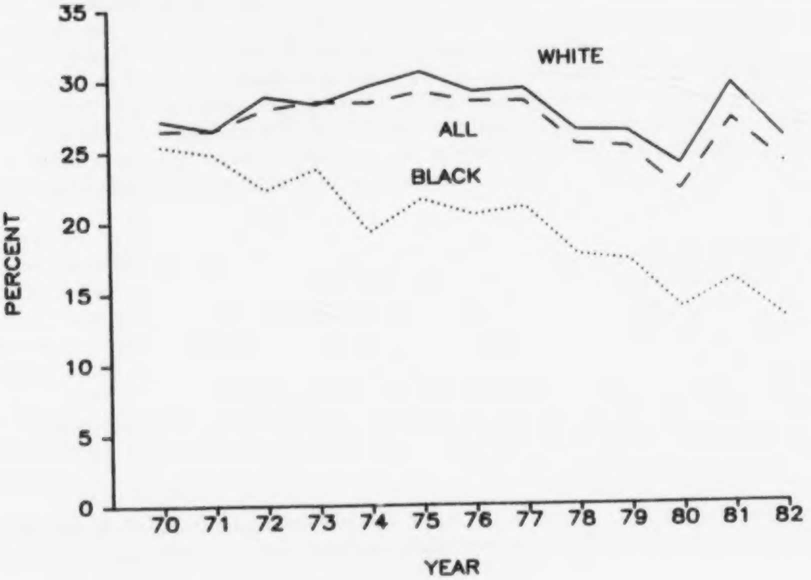


During 1979-1980, differences in hysterectomy rates between blacks and whites declined noticeably. In 1981, however, the rates of blacks and whites diverged, only to converge again in 1982. The instability of race-specific rates may be due, in part, to the two different methods of imputing unknowns for race in 1981 and 1982. Similarly, the divergence in percentages of vaginal hysterectomies between blacks and whites that was seen in the period 1981-1982 may be due to unstable race-specific hysterectomy rates, which, in turn, may be due either to the different methods of redistributing unknowns for race or to the small absolute number of hysterectomies among blacks surveyed.

Variations in hysterectomy rates by region have persisted since 1970 (*2-4*). Factors related to both patients and physicians may contribute to these regional differences. Patient-related factors may include regional differences in the incidence of gynecologic conditions and in general attitudes toward surgery or sterilization during the reproductive years (*7*). Physician-related factors may include regional differences in trends in training and practice (*8,9*).

In conclusion, while 1982 national and regional hysterectomy rates reached their lowest level since 1970, those for the period 1981-1982 may represent either the beginning of a plateau or a continued decline in rates since 1975. Further surveillance of hysterectomy rates among women of reproductive age in U.S. non-Federal, short-stay hospitals will determine the direction of these trends in the early 1980s.

FIGURE 5. Vaginal hysterectomies as a percentage of all hysterectomies for women ages 15-44 years, by race, United States, 1970-1982



References

1. National Center for Health Statistics. Detailed diagnoses and surgical procedures for patients discharged from short-stay hospitals: United States, 1979. Hyattsville, Maryland: National Center for Health Statistics, 1982; DHHS publication no. (PHS) 82-1274-1.
2. CDC. Surgical sterilization surveillance; hysterectomy in women aged 15-44, 1970-1975. Atlanta, Georgia: Centers for Disease Control, 1980.
3. CDC. Surgical sterilization surveillance; hysterectomy in women aged 15-44, 1976-1978. Atlanta, Georgia: Centers for Disease Control, 1981.
4. CDC. Hysterectomy among women of reproductive age, United States, update for 1979-1980. In: CDC Surveillance Summaries (published four times a year). August 1983; 32 (No 3SS):1SS-7SS.
5. National Center for Health Statistics. Utilization of short-stay hospitals: United States, 1981, annual summary. Hyattsville, Maryland: National Center for Health Statistics, 1983; DHHS publication no. (PHS)83-1733. (Vital and health statistics; series 13; no.72).
6. Nolan TF, Ory HW, Layde PM, Hughes JM, Greenspan JR. Cumulative prevalence rates and corrected incidence rates of surgical sterilization among women in the United States, 1971-1978. Am J Epidemiol 1982;116:776-81.
7. Rochat RW. Regional variation of sterility, United States, 1970. Advances in Planned Parenthood 1976;9:1-11.
8. Wennberg JE, Gittlesohn A. Variations in medical care among small areas. Scientific American 1982;246:120-34.
9. Wennberg JE. Factors governing utilization of hospital services. Hosp Pract 1979;14:115-27.

Foodborne Disease Outbreaks, Annual Summary, 1982

Kristine L. MacDonald, M.D.
Patricia M. Griffin, M.D.
Enteric Diseases Branch
Division of Bacterial Diseases
Center for Infectious Diseases

Introduction

Foodborne disease surveillance has traditionally served three objectives:

1. **Disease Prevention and Control**. Early identification and removal of contaminated products from the commercial market, correction of faulty food preparation practices in food service establishments and in the home, and identification and appropriate treatment of human carriers of foodborne pathogens are the main prevention and control measures that result from surveillance of foodborne disease.

2. **Knowledge of Disease Causation**. In over half of the foodborne disease outbreaks reported to CDC in each of the last 5 years, the responsible pathogen was not identified. In many of these outbreaks, pathogens known to cause foodborne illness may not have been identified because laboratory investigations were late or incomplete. In others, the responsible pathogen may have escaped detection even after thorough laboratory investigation, either because the pathogen may not have been recognized as a cause of foodborne disease or because the pathogen could not be identified by available laboratory techniques. When more thorough clinical, epidemiologic, and laboratory investigations are conducted, perhaps many of these pathogens can be identified, and suitable measures for prevention and control can be instituted.

3. **Administrative Guidance**. The collection of data from investigations of foodborne disease outbreaks permits the assessment of trends in the prevalence of etiologic agents and in vehicles of disease transmission. In addition, it brings to light common errors in food handling. Compiling and publishing annual data will enable local and state health departments and others involved in the implementation of food protection programs to be kept informed of the factors involved in foodborne disease outbreaks. Comprehensive surveillance should result in a greater awareness of the most important food protection methods, the institution of better training programs, and more effective use of available resources.

Methods

Definition of Outbreak. For the purpose of this report, a foodborne disease outbreak was defined as an incident in which 1) two or more persons experience a similar illness after ingestion of a common food, and 2) epidemiologic analysis implicates the food as the source of the illness. A few exceptions exist; for example, one case of botulism or chemical poisoning constitutes an outbreak.

Outbreaks of known etiology are those for which laboratory evidence of a specific agent is obtained and specified criteria are met. Outbreaks of unknown etiology are those for which epidemiologic evidence implicates a food source, but adequate laboratory confirmation is not obtained.

Source of Data. Outbreaks are reported to CDC on a standard reporting form. Reports come most frequently from state and local health departments; they may also be received from Federal agencies such as the Food and Drug Administration (FDA), the U.S. Department of Agriculture (USDA), the U.S. Armed Forces, and occasionally from private physicians. Forms are reviewed at CDC to determine whether a specific etiologic agent for the outbreak can be confirmed and, in some instances, questions about an etiologic agent may be referred back to the reporting agency. Otherwise, data are accepted as reported.

Interpretation of Data. The limitations on the quantity and quality of data presented here must be recognized in order to avoid misinterpretation. The number of outbreaks of foodborne disease reported by this surveillance system clearly represents only a small fraction of the total number that occur. The likelihood of an outbreak's coming to the attention of health authorities is dependent on consumers' and physicians' awareness, their interest, and their motivation to report the incident. For example, large outbreaks; interstate outbreaks; restaurant-associated outbreaks; and outbreaks involving serious illnesses, hospitalizations, or deaths are more likely to come to the attention of health authorities than cases of mild illness following a family cookout.

The quality of the data presented here depends upon the commitment to surveillance of this type of disease by state or local health departments. A department's interest in foodborne disease, its investigative capabilities, and its laboratory facilities are important determinants of the quality of the investigation. Furthermore, the likelihood that the findings of the investigation will be reported varies from one locality to another. This report, then, should not be the basis of firm conclusions about the absolute incidence of foodborne disease, and it should not be used to draw conclusions about the relative incidence of foodborne diseases by specific causes. For example, foodborne diseases characterized by short incubation periods, such as those caused by a chemical agent or staphylococcal enterotoxin, are more likely to be recognized in common-source foodborne disease outbreaks than those diseases with longer incubation periods, such as hepatitis A. Outbreaks involving less common pathogens, such as *Bacillus cereus*, *Escherichia coli*, or *Giardia lamblia* are less likely to be confirmed because these organisms are often not considered in clinical, epidemiologic, and laboratory investigations of foodborne disease outbreaks. Also, since laboratory methods for confirming viral disease are not widely available, many outbreaks of foodborne illness due to viruses probably fall into the "unknown etiologic agent" category. Pathogens that generally cause mild illness will be underrepresented, while those causing serious illness, such as *Clostridium botulinum*, are more likely to be identified. Similarly, restaurant- or commercial-product-associated outbreaks have a higher likelihood of being reported.

Results

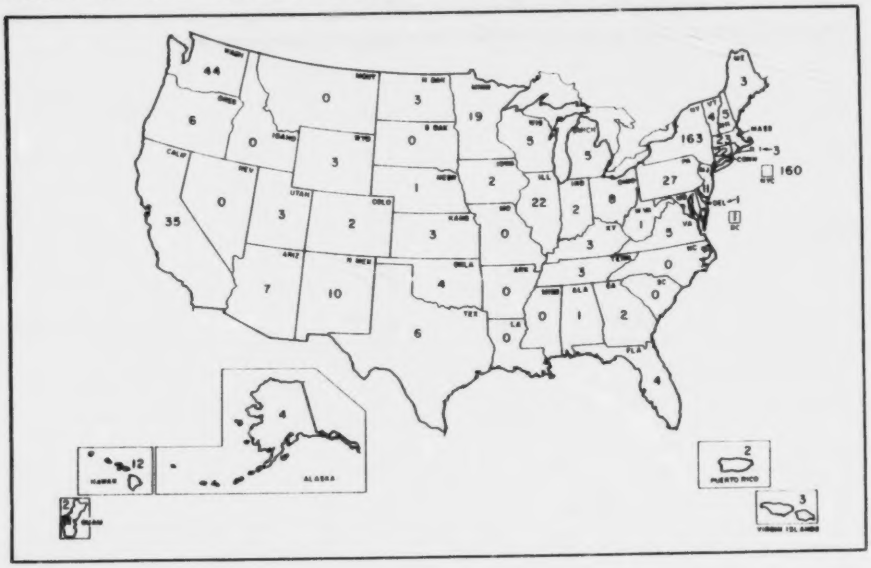
In 1982, 656 outbreaks (19,380 cases) of foodborne disease were reported to CDC. Reports were received from 39 states as well as from the District of Columbia, the U.S. Virgin Islands, Guam, and Puerto Rico (Figure 1). New York reported the largest number of outbreaks (323), with 160 from New York City; Washington reported the next largest number of outbreaks (44), followed by California (35).

The etiologic agent was confirmed in 34% of the outbreaks (11,050 cases) (Table 1). Bacterial pathogens accounted for 151 outbreaks (5,501 cases). The most frequently isolated bacterial pathogen was *Salmonella* (55 outbreaks, 2,056 cases), followed by *Staphylococcus aureus* (28 outbreaks, 669 cases) and *Clostridium perfringens* (22 outbreaks, 1,189 cases). During 1982, two outbreaks (one in Oregon and one in Michigan) of a previously unrecognized pathogen, *E. coli* O157:H7, were investigated. Both outbreaks were associated with eating hamburger from the same fast-food restaurant chain. The illness was characterized by bloody diarrhea, abdominal cramps, and low-grade or absent fever. Since it was first described, this pathogen has also been associated with non-bloody diarrhea and a spectrum of clinical illnesses, including hemolytic-uremic syndrome. Viral agents (hepatitis A and Norwalk virus) accounted for 21 outbreaks (5,325 cases). The high number of cases resulting from viral agents is predominantly due to two large outbreaks of Norwalk gastroenteritis that occurred in Minnesota. One outbreak involved 3,000 cases and was related to eating bakery items with frosting. The second involved 2,000 cases and was associated with eating cole slaw. Chemical agents were responsible for 47 outbreaks (220 cases). Only one outbreak, involving four persons with *Trichinella spiralis,* was attributed to a parasitic agent. Twenty-six deaths were associated with foodborne diseases in 1982: 11 from *Vibrio cholerae* O1 (all in Guam and the Trust Territories), eight from *Salmonella,* five from *C. botulinum,* and two from unknown causes. No pathogen was identified in 436 of the outbreaks (8,330 cases) reported in 1982.

Incubation periods were known for illnesses in 412 of the outbreaks. In 13 outbreaks, the incubation period was less than 1 hour; in 123 outbreaks, it was 1-7 hours; in 91 outbreaks, it was 8-14 hours; and in 185 outbreaks, it was 15 hours or more.

The food vehicle of transmission was determined in 168 (76%) of the 220 outbreaks in which a pathogen was identified (Table 2). For diseases caused by bacterial pathogens, meat or dairy products were the vehicles of transmission in 52% of the outbreaks in which the vehicle was known. For diseases caused by chemicals, fish or shellfish were the vehicles of transmission in 57% of the outbreaks. Food eaten in a restaurant was associated with 44% of outbreaks, and food eaten at home, with 30% (Table 3). The proportion of foodborne illness due to known etiologic agents was higher in the summer months than at any other time of year (Table 4).

FIGURE 1. Outbreaks of foodborne disease reported to CDC, by state, 1982



WASH 44
OREG 6
IDAHO 0
MONT 0
WYO 3
N DAK 3
S DAK 0
MINN 19
WIS 5
MICH 5
ME 3
NY 163
VT 4
NH 5
MASS
CONN
R I—3
NYC 160
CALIF 35
NEV 0
UTAH 3
COLO 2
NEBR 1
IOWA 2
ILL 22
IND 2
OHIO 8
PA 27
DEL—1
DC 1
KANS 3
MO 0
KY 3
W VA 1
VA 5
ARIZ 7
N MEX 10
OKLA 4
ARK 0
TENN 3
NC 0
SC 0
TEX 6
LA 0
MISS 0
ALA 1
GA 2
FLA 4
ALASKA 4
HAWAII 12
GUAM 2
PUERTO RICO 2
VIRGIN ISLANDS 3

Discussion

The inconsistency in reporting can be seen by looking at the distribution of outbreaks by state. A few states, such as New York and Washington, reported a disproportionately large number of outbreaks. For example, New York State and New York City reported 323 outbreaks, almost one-half of those reported for the entire United States. While it is possible that states such as New York and Washington have a higher rate of foodborne disease, it is more likely that these figures simply represent differences in surveillance activity. The same inconsistency can be seen when looking at outbreaks by pathogen. CDC data show that *C.botulinum* is as common a foodborne pathogen as *Campylobacter jejuni* and *Vibrio parahaemolyticus*, a finding that indicates only that botulism is reported more comprehensively than some other pathogens.

The number of outbreaks of foodborne disease of confirmed etiology that were reported to CDC over the last 5 years has remained relatively constant. The distribution of cases by etiologic agent has also remained fairly consistent. Etiologic agents typically have been confirmed in about 40% of all outbreaks. When etiologic agents have been confirmed, bacterial pathogens have consistently accounted for approximately two-thirds of the outbreaks, and chemical agents for an additional 20%-25%.

Many food preparation practices contribute to foodborne disease (Table 5). In 1982, the five most common practices, in order of frequency of occurrence, included 1) improper holding temperature, 2) food from an unsafe source, 3) inadequate cooking, 4) poor personal hygiene on the part of food handlers, and 5) contaminated equipment. In most of the outbreaks caused by bacterial pathogens, the food had been stored at improper holding temperatures. In outbreaks of botulism or trichinosis, the food had usually been inadequately cooked. In outbreaks of ciguatera and mushroom poisoning, the food itself was unsafe, and illness was not related to improper handling or preparation.

The large number of outbreaks in which no pathogen was identified points out the need for the improvement of investigative skills so that known pathogens can be identified more frequently, and new and as-yet-unidentified pathogens can be recognized.

TABLE 1. Confirmed foodborne disease outbreaks, cases, and deaths, by etiologic agents, United States, 1982

Etiologic agent	Outbreaks No.	Outbreaks (%)	Cases No.	Cases (%)	Deaths No.	Deaths (%)
BACTERIAL						
Bacillus cereus	8	(3.6)	200	(1.8)	0	(0.0)
Brucella	1	(0.5)	3	(< 0.1)	0	(0.0)
Campylobacter jejuni	2	(0.9)	31	(0.3)	0	(0.0)
Clostridium botulinum	21	(9.5)	30	(0.3)	5	(20.9)
Clostridium perfringens	22	(10.0)	1,189	(10.8)	0	(0.0)
Escherichia coli	2	(0.9)	47	(0.4)	0	(0.0)
Salmonella	55	(25.0)	2,056	(18.6)	8	(33.3)
Shigella	4	(1.8)	116	(1.1)	0	(0.0)
Staphylococcus aureus	28	(12.7)	669	(6.0)	0	(0.0)
Streptococcus Group A	1	(0.5)	34	(0.3)	0	(0.0)
Vibrio cholerae O1	1	(0.5)	892	(8.0)	11	(45.8)
Vibrio cholerae non-O1	1	(0.5)	7	(0.1)	0	(0.0)
Vibrio parahaemolyticus	3	(1.4)	39	(0.4)	0	(0.0)
Yersinia enterocolitica	2	(0.9)	188	(1.7)	0	(0.0)
Total	151	(68.7)	5,501	(49.9)	24	(100.0)
CHEMICAL						
Ciguatoxin	8	(3.6)	37	(0.3)	0	(0.0)
Heavy metals	5	(2.3)	26	(0.2)	0	(0.0)
Monosodium glutamate	3	(1.4)	10	(0.1)	0	(0.0)
Mushrooms	4	(1.8)	9	(0.1)	0	(0.0)
Scombrotoxin	18	(8.2)	58	(0.5)	0	(0.0)
Shellfish	1	(0.5)	5	(< 0.1)	0	(0.0)
Other	8	(3.6)	75	(0.7)	0	(0.0)
Total	47	(21.4)	220	(1.9)	0	(0.0)
PARASITIC						
Trichinella spiralis	1	(0.5)	4	(< 0.1)	0	(0.0)
Total	1	(0.5)	4	(< 0.1)	0	(0.0)
VIRAL						
Hepatitis A	19	(8.5)	325	(2.9)	0	(0.0)
Norwalk virus	2	(0.9)	5,000	(45.2)	0	(0.0)
Total	21	(9.4)	5,325	(48.1)	0	(0.0)
CONFIRMED TOTAL	**220**	**(100.0)**	**11,050**	**(100.0)**	**24**	**(100.0)**

TABLE 2. Foodborne outbreaks by specific etiologic

Etiologic agent	Beef	Ham	Pork	Sausage	Chicken	Turkey	Other meat	Shellfish	Tuna	Mahi-Mahi	Other fish	Milk	Eggs	Ice cream
BACTERIAL														
B. cereus	1	-	-	-	-	-	-	-	-	-	-	-	-	1
Brucella	-	-	-	-	-	-	-	-	-	-	-	-	-	-
C. jejuni	-	-	-	-	-	-	-	-	-	-	-	2	-	-
C. botulinum	1	-	-	-	-	-	-	-	-	-	1	-	-	-
C. perfringens	6	-	-	-	-	4	2	-	-	-	-	-	-	-
E. coli	2	-	-	-	-	-	-	-	-	-	-	-	-	-
Salmonella	1	-	2	1	2	4	1	-	-	-	1	1	2	4
Shigella	-	-	-	-	-	-	-	-	1	-	-	-	-	-
S. aureus	5	3	2	1	4	3	1	-	-	-	-	-	-	-
Streptococcus Group A	-	-	-	-	-	-	-	-	-	-	-	-	-	-
V. cholerae O1	-	-	-	-	-	-	-	-	-	-	-	-	-	-
V. cholerae non-O1	-	-	-	-	-	-	-	1	-	-	-	-	-	-
V. parahaemolyticus	-	-	-	-	-	-	-	3	-	-	-	-	-	-
Y. enterocolitica	-	-	-	-	-	-	-	-	-	-	-	1	-	-
Other	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Total	16	3	4	2	6	11	4	4	1	-	2	4	2	5
CHEMICAL														
Ciguatoxin	-	-	-	-	-	-	-	-	-	-	8	-	-	-
Heavy metals	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Monosodium glutamate	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Mushrooms	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Shellfish	-	-	-	-	-	-	-	1	-	-	-	-	-	-
Scombrotoxin	-	-	-	-	-	-	-	-	3	5	10	-	-	-
Other	1	-	-	1	-	-	-	-	-	-	-	-	-	-
Total	1	-	-	1	-	-	-	1	3	5	18	-	-	-
PARASITIC														
Trichinella spiralis	-	-	1	-	-	-	-	-	-	-	-	-	-	-
Total	-	-	1	-	-	-	-	-	-	-	-	-	-	-
VIRAL														
Hepatitis A	-	-	-	-	-	-	-	5	1	-	-	-	-	1
Norwalk virus	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Total	-	-	-	-	-	-	-	5	1	-	-	-	-	1
CONFIRMED TOTAL	17	3	5	3	6	11	4	10	5	5	20	4	2	6
UNKNOWN	5	2	1	1	7	-	-	56	2	-	1	-	1	-
TOTAL 1982	22	5	6	4	13	11	4	66	7	5	21	4	3	6

agent and vehicle of transmission, United States, 1982

Baked foods	Fruits & vegetable	Potato salad	Poultry, fish, egg salad	Other salad	Fried rice	Chinese food	Mexican food	Carbonated bev	Nondairy bev	Multiple foods	Mushrooms	Other foods	Unknown	Total
1	-	-	-	1	3	-	-	-	-	-	-	1	-	8
-	-	-	-	-	-	-	-	-	-	-	-	-	1	1
-	-	-	-	-	-	-	-	-	-	-	-	-	-	2
-	16	-	-	-	-	-	-	-	-	-	1	1	1	21
-	-	-	-	-	-	-	2	-	-	1	-	-	7	22
-	-	-	-	-	-	-	-	-	-	-	-	-	-	2
-	-	-	1	2	-	-	1	-	-	-	-	3	29	55
-	-	-	-	1	-	-	-	-	-	-	-	-	2	4
2	-	2	1	-	-	-	1	-	-	1	-	-	2	28
-	-	-	-	1	-	-	-	-	-	-	-	-		1
-	-	-	-	-	-	-	-	-	-	1	-	-	-	1
-	-	-	-	-	-	-	-	-	-	-	-	-	-	1
-	-	-	-	-	-	-	-	-	-	-	-	-	-	3
-	1	-	-	-	-	-	-	-	-	-	-	-	-	2
-	-	-	-	-	-	-	-	-	-	-	-	-		
3	17	2	2	5	3	-	4		-	3	1	5	42	151
-	-	-	-	-	-	-	-	-	-	-	-	-	-	8
-	-	-	-	-	-	-	-	4	1	-	-	-	-	5
-	-	-	-	-	-	2	-	-	-	1	-	-	-	3
-	-	-	-	-	-	-	-	-	-	-	4	-	-	4
-	-	-	-	-	-	-	-	-	-	-	-	-	-	1
-	-	-	-	-	-	-	-	-	-	-	-	-	-	18
	1	-	-	-	-	-	-	1	1	-	-	3	-	8
-	1	-	-	-	-	2	-	5	2	1	4	3	-	47
-	-	-	-	-	-	-	-	-	-	-	-	-		1
-	-	-	-	-	-	-	-	-	-		-	-		1
1	-	-	-	-	-	-	-	-		1	-	-	10	19
1	-	-	-	1	-	-	-	-	-	-	-	-	-	2
2	-	-	-	1	-	-	-	-	-	1	-	-	10	21
5	18	2	2	6	3	2	4	5	2	5	5	8	52	220
5	1	3	1	6	1	1	2	-	-	5	1	9	325	436
10	19	5	3	12	4	3	6	5	2	10	6	17	377	656

TABLE 3. Foodborne disease outbreaks, by specific etiologic agent and place where food was eaten, United States, 1982

Etiologic agent	Home	Delicatessen, cafeteria, or restaurant	School	Picnic	Church	Camp	Other	Unknown	Total
BACTERIAL									
B. cereus	-	3	2	-	-	1	2	-	8
Brucella	1	-	-	-	-	-	-	-	1
C. jejuni	1	-	-	-	-	-	1	-	2
C. botulinum	21	-	-	-	-	-	-	-	21
C. perfringens	-	9	1	-	-	-	12	-	22
E. coli	-	-	-	-	-	-	2	-	2
Salmonella	12	17	6	3	4	1	12	-	55
Shigella	1	2	-	-	1	-	-	-	4
S. aureus	4	7	2	2	2	1	10	-	28
Streptococcus Group A	-	-	-	-	-	-	1	-	1
V. cholerae O1	1	-	-	-	-	-	-	-	1
V. cholerae non-O1	1	-	-	-	-	-	-	-	1
V. parahaemolyticus	1	-	-	1	-	-	1	-	3
Y. enterocolitica	1	-	-	-	-	-	1	-	2
Total	44	38	11	6	7	3	42	-	151
CHEMICAL									
Ciguatoxin	3	5	-	-	-	-	-	-	8
Heavy metals	1	3	-	-	-	-	1	-	5
Monosodium glutamate	1	2	-	-	-	-	-	-	3
Mushroom	3	-	-	-	-	-	1	-	4
Shellfish	1	-	-	-	-	-	-	-	1
Scombrotoxin	7	9	-	-	-	-	2	-	18
Other chemical	4	4	-	-	-	-	-	-	8
Total	20	23	-	-	-	-	4	-	47
PARASITIC									
Trichinella spiralis	1	-	-	-	-	-	-	-	1
Total	1	-	-	-	-	-	-	-	1
VIRAL									
Hepatitis A	5	6	-	1	-	1	6	-	19
Norwalk virus	-	1	-	-	-	-	1	-	2
Total	5	7	-	1	-	1	7	-	21
CONFIRMED TOTAL	70	68	11	7	7	4	53	-	220
UNKNOWN	127	221	17	16	2	1	49	3	436
TOTAL 1982	**197**	**289**	**28**	**23**	**9**	**5**	**102**	**3**	**656**

TABLE 4. Foodborne disease outbreaks by specific etiologic agent and month of occurrence, United States, 1982

Etiologic agent	Jan	Feb	Mar	Apr	May	Jun	Jul	Aug	Sep	Oct	Nov	Dec	Unknown	Total
BACTERIAL														
Brucella	-	-	1	-	-	-	-	-	-	-	-	-	-	1
B. cereus	2	-	-	-	-	-	2	2	2	-	-	-	-	8
C. jejuni	-	-	-	-	-	1	-	-	1	-	-	-	-	2
C. botulinum	-	3	-	-	2	3	3	2	1	1	3	3	-	21
C. perfringens	-	2	1	1	5	2	1	2	2	1	2	3	-	22
E. coli	-	1	-	-	1	-	-	-	-	-	-	-	-	2
Salmonella	2	1	1	5	4	5	10	11	3	5	3	5	-	55
Shigella	-	-	-	1	-	-	-	-	1	1	1	-	-	4
S. aureus	2	1	1	2	2	3	5	5	1	3	1	2	-	28
Streptococcus Group A	-	-	-	-	-	-	1	-	-	-	-	-	-	1
V. cholerae O1	-	-	-	-	-	-	-	1	-	-	-	-	-	1
V. cholerae non-O1	-	-	-	-	-	-	-	-	-	-	-	1	-	1
V. parahaemolyticus	-	-	-	-	-	-	2	1	-	-	-	-	-	3
Y. enterocolitica	-	1	-	-	-	1	-	-	-	-	-	-	-	2
Total	6	9	4	9	14	15	24	24	11	11	10	14	-	151
CHEMICAL														
Ciguatoxin	1	-	-	-	-	-	3	3	-	-	1	-	-	8
Heavy metals	1	1	-	-	1	-	-	-	-	-	2	-	-	5
Monosodium glutamate	1	-	1	-	-	-	-	1	-	-	-	-	-	3
Mushrooms	-	-	1	-	-	-	2	-	1	-	-	-	-	4
Shellfish	-	-	-	-	-	-	-	1	-	-	-	-	-	1
Scombrotoxin	1	-	-	-	-	1	1	4	6	4	-	1	-	18
Other	-	-	2	2	1	-	-	-	1	1	1	-	-	8
Total	4	1	4	2	2	1	6	9	8	5	4	1	-	47
PARASITIC														
Trichinella spiralis	-	-	-	-	1	-	-	-	-	-	-	-	-	1
Total	-	-	-	-	1	-	-	-	-	-	-	-	-	1
VIRAL														
Hepatitis A	3	-	-	-	2	3	3	4	1	1	-	2	-	19
Norwalk virus	-	-	-	-	-	-	-	1	-	-	1	-	-	2
Total	3	-	-	-	2	3	3	5	1	1	1	2	-	21
CONFIRMED TOTAL	13	10	8	11	19	19	33	38	20	17	15	17	-	220
UNKNOWN	27	31	27	29	43	31	21	32	30	27	26	112	-	436
TOTAL 1982	**40**	**41**	**35**	**40**	**62**	**50**	**54**	**70**	**50**	**44**	**41**	**129**	-	**656**

TABLE 5. Foodborne disease outbreaks by etiologic agent and contributing factors, United States, 1982

Etiologic agent	Number of reported outbreaks	Outbreaks in which factors reported	Improper holding temperatures	Inadequate cooking	Contaminated equipment	Food from unsafe source	Poor personal hygiene	Other
BACTERIAL								
B. cereus	8	5	5	-	-	-	1	-
Brucella	1	-	-	-	-	-	-	-
C. jejuni	2	2	-	-	-	1	-	1
C. botulinum	21	4	1	-	-	-	-	3
C. perfringens	22	20	19	8	3	-	-	2
E. coli	2	1	1	-	-	-	-	-
Salmonella	55	34	16	18	6	6	7	4
Shigella	4	4	1	-	-	-	4	-
S. aureus	28	21	20	3	4	1	9	1
Streptococcus Group A	1	1	1	-	-	-	1	-
Streptococcus Group D	1	-	-	-	-	-	-	-
V. cholerae O1	1	-	-	-	-	-	-	-
V. cholerae non-O1	1	1	-	1	-	-	-	-
V. parahaemolyticus	3	2	-	-	-	2	-	-
Y. enterocolitica	2	-	-	-	-	-	-	-
Total	151	95	64	30	13	10	22	11
CHEMICAL								
Ciguatoxin	8	1	-	-	-	1	-	-
Heavy metals	5	4	-	-	2	-	1	2
Monosodium glutamate	3	2	-	-	-	-	-	2
Mushrooms	4	2	-	-	-	2	-	-
Shellfish	1	1	-	-	-	1	-	-
Scombrotoxin	18	9	5	-	-	5	-	-
Other	8	6	-	-	3	-	-	3
Total	47	25	5	-	5	9	1	7
PARASITIC								
Trichinella spiralis	1	1	-	1	-	1	-	-
Total	1	1	-	1	-	1	-	-
VIRAL								
Hepatitis A	19	14	2	2	-	6	9	3
Norwalk virus	2	2	-	-	-	-	2	-
Total	21	16	2	2	-	6	11	3
CONFIRMED TOTAL	220	137	71	33	18	26	34	21
UNKNOWN	436	225	87	73	35	94	59	16
TOTAL 1982	**656**	**362**	**158**	**106**	**53**	**120**	**93**	**37**

Nosocomial Infection Surveillance, 1984

Teresa C. Horan, M.P.H.
John W. White, Ph.D.
William R. Jarvis, M.D.
T. Grace Emori, R.N., M.S.
David H. Culver, Ph.D.
Van P. Munn, B.S.
Clyde Thornsberry, Ph.D.
David R. Olson, Ph.D.
James M. Hughes, M.D.
Hospital Infections Program
Center for Infectious Diseases

Introduction

Nosocomial infections cause substantial morbidity and mortality, prolong the hospital stay of affected patients, and increase direct patient-care costs (*1-5*). Since 1970, the National Nosocomial Infections Surveillance System (NNIS) has collected and analyzed data on the frequency of nosocomial infections in U.S. hospitals. This report provides descriptive data on nosocomial infections in a sample of U.S. hospitals in 1984.

Materials and Methods

The methods used in this surveillance system and the characteristics of participating hospitals have been described in detail (*4,6*). In brief, hospitals participating in NNIS conduct active hospital-wide surveillance using uniform definitions of nosocomial infections. Although the definitions are specific for different sites of infection, onset must occur during hospitalization or shortly after discharge, and the infection may not be present or incubating at the time of the patient's admission. Each month data are recorded on standardized forms that are sent to CDC, where they are coded, edited, and entered into a computer before being analyzed. In 1984, 51 hospitals regularly (≥ 9 months) reported data to CDC. For each nosocomial infection detected, the following information was reported: site of infection; date of onset; hospital service on which the patient was placed; age and sex of the patient; pathogens isolated; occurrence of secondary bacteremia; antimicrobial susceptibility of bacterial pathogens; and, for those patients who died with a nosocomial infection, the relationship of the infection to death. In addition, the hospitals reported the number of patients discharged each month from six primary services: medicine, surgery, obstetrics, gynecology, pediatrics, and newborn.

Results

The NNIS Sample. The hospitals participating in NNIS are not a probability sample of U.S. hospitals; however, those hospitals that regularly reported data in 1984 ranged in size from 80 to 1,200 beds, were located throughout the United States, and were owned by state and local governments, as well as by profit and nonprofit organizations. The geographic distribution of the 51 hospitals among the four regions of the country (Northeast, North Central, South, and West) was roughly the same as that for all 6,375 U.S. acute-care hospitals included in the American Hospital Association Annual Survey of Hospitals (*7*). Hospitals affiliated with medical schools, referred to as teaching hospitals, are still greatly overrepresented among the NNIS hospitals; 61% (31/51) of the NNIS hospitals are teaching hospitals, whereas only 17% of the hospitals across the country are affiliated with a medical school. Similarly, the 51 NNIS hospitals tend to be large, with a median size of 406 beds, compared with a median size of only 112 beds for the 5,375 U.S. acute-care hospitals (*7*).

Despite these limitations, previous analyses have shown that data collected in NNIS can be usefully interpreted by stratifying the 51 reporting hospitals into three groups: 1) 20 (39%) nonteaching hospitals, 2) 18 (35%) small teaching hospitals of 500 or fewer beds, and 3) 13 (26%) large teaching hospitals of more than 500 beds (*4,6*).

The overall infection rate (number of hospital-acquired infections per 1,000 patients discharged) was highest in the large teaching hospitals and lowest in the nonteaching hospitals (Table 1). In all three hospital categories, the infection rate was highest on the surgery service, followed by the medicine and gynecology services (Table 2). On each of the six primary services, the infection rate was highest at the large teaching hospitals and lowest at the nonteaching hospitals, with the exception of the gynecology service rate, which was highest at small teaching hospitals.

In all three hospital categories, the urinary tract was the site most frequently infected, followed by lower respiratory tract or surgical wound infections (Table 3). For each site of infection, the infection rates were highest in the large teaching hospitals and lowest in the nonteaching hospitals.

Infections of the urinary tract, of surgical wounds, and of the lower respiratory tract accounted for almost three-fourths of the infections in all three hospital categories (Table 4). Primary bacteremia and cutaneous infections accounted for a higher percentage of infections in the large teaching hospitals than in the other hospitals.

Combined Rates by Service and Site. In general, the site-specific infection rate on each service was highest in the large teaching hospitals and lowest in the nonteaching hospitals

TABLE 1. Infection rates (cases/1,000 discharges), by hospital category, 1984

Hospital category	Infections	Discharges	Rate
Nonteaching	4,960	223,909	22.2
Small teaching	9,031	267,078	33.8
Large teaching	12,974	313,697	41.4
Total	**26,965**	**804,684**	**33.5**

TABLE 2. Infection rates (cases/1,000 discharges), by hospital category and service, 1984

	Service*					
Hospital category	SURG	MED	GYN	OB	NEW	PED
Nonteaching	30.8	23.3	8.6	5.6	8.6	1.2
Small teaching	47.3	38.1	35.2	14.9	14.7	14.6
Large teaching	59.3	46.9	31.7	20.3	17.3	16.6
Total	**46.7**	**36.5**	**28.1**	**15.3**	**14.4**	**13.3**

*SURG = surgery, MED = medicine, GYN = gynecology, OB = obstetrics, NEW = newborn, PED = pediatrics

(Table 5). In each hospital category, urinary tract infections occurred predominantly on the medicine, surgery, and gynecology services. Surgical wound infections occurred primarily on the surgery, gynecology, and obstetrics services. Lower respiratory infections occurred predominantly on the surgery and medicine services. Primary bacteremia occurred most frequently on the medicine and surgery services at nonteaching and large teaching hospitals. At small teaching hospitals, primary bacteremia was most frequently seen on the medicine and pediatrics services, followed by the newborn and surgery services. Cutaneous infections occurred primarily on the newborn service in each hospital category.

Pathogens. Of the 26,965 infections reported, 64% were caused by single pathogens, and 20% were caused by multiple pathogens (Figure 1). No pathogen was identified in 6% of the infections, and no culture was obtained in 10%. Of the 84% of infections in which a pathogen was identified, 86% were caused by aerobic bacteria, 2% by anaerobic bacteria, and 8% by fungi. Viruses, protozoa, and parasites collectively accounted for 5% of the infections of known etiology.

Escherichia coli, Pseudomonas aeruginosa, enterococci, and *Staphylococcus aureus* were the most frequently reported pathogens (Table 6). *E. coli* was the pathogen most often reported on all services except pediatrics and newborn, where *S. aureus* was the most common. *P. aeruginosa* was the second most frequently identified pathogen on the medicine and surgery services, whereas enterococci were second on the gynecology and obstetrics services. Coagulase-negative staphylococci were the second most frequently identified pathogens on the pediatrics and newborn services.

TABLE 3. Infection rates (cases/1,000 discharges), by hospital category and site of infection, 1984

	Site*					
Hospital category	**UTI**	**SWI**	**LRI**	**BACT**	**CUT**	**Other**
Nonteaching	9.9	3.6	4.2	1.3	1.1	2.0
Small teaching	13.9	6.0	5.4	1.9	1.8	4.7
Large teaching	14.2	6.6	7.7	3.9	2.6	6.4
Total	**12.9**	**5.6**	**6.0**	**2.5**	**1.9**	**4.6**

*UTI = urinary tract infection, SWI = surgical wound infection, LRI = lower respiratory infection, BACT = primary bacteremia, CUT = cutaneous infection

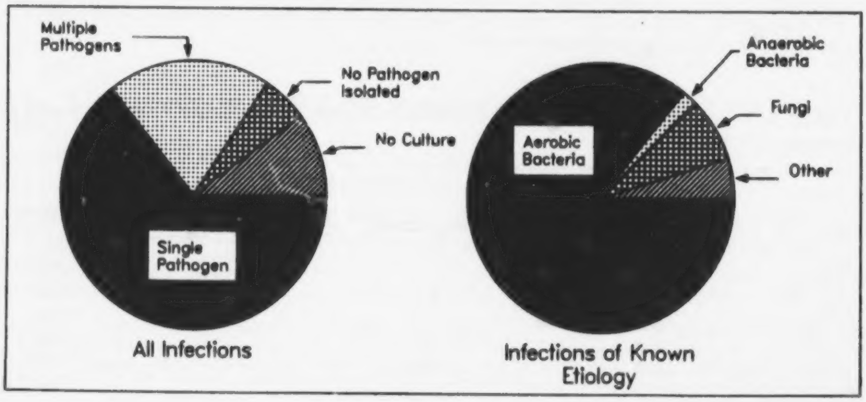
TABLE 4. Percentage distribution of infections at each of the major sites, by hospital category, 1984

	Hospital category			
Site	**Nonteaching**	**Small teaching**	**Large teaching**	**Total**
UTI	44.6	41.2	34.2	**38.5**
SWI	16.0	17.8	16.0	**16.6**
LRI	19.2	15.9	18.6	**17.8**
BACT	6.0	5.7	9.4	**7.5**
CUT	4.9	5.4	6.3	**5.8**
Other	9.3	14.0	15.4	**13.8**

TABLE 5. Site-specific infection rates (cases/1,000 discharges), by service, 1984

Service	Site						
	UTI	SWI	LRI	BACT	CUT	Other	All sites
1. Nonteaching hospitals							
SURG	12.1	8.5	5.4	1.3	1.4	2.0	30.8
MED	12.6	0.4	5.2	1.9	0.8	2.3	23.3
GYN	5.8	1.6	0.1	0.1	0.1	0.8	8.6
OB	1.1	2.4	0.1	0.1	0.5	1.4	5.6
PED	0.0	0.1	0.1	0.0	0.1	0.9	1.2
NEW	0.5	0.2	1.8	0.6	2.6	2.9	8.6
Total	**9.9**	**3.6**	**4.2**	**1.3**	**1.1**	**2.0**	**22.2**
2. Small teaching hospitals							
SURG	17.7	13.6	7.8	1.8	1.6	4.7	47.3
MED	20.1	0.6	7.5	2.8	1.7	5.3	38.1
GYN	19.9	11.1	1.3	0.4	0.2	2.2	35.2
OB	3.8	6.8	0.3	0.2	0.5	3.4	14.9
PED	2.0	0.6	2.0	2.4	2.3	5.2	14.6
NEW	0.6	0.2	1.4	2.0	4.8	5.6	14.7
Total	**13.9**	**6.0**	**5.4**	**1.9**	**1.8**	**4.7**	**33.8**
3. Large teaching hospitals							
SURG	19.5	15.0	11.2	4.2	3.3	6.1	59.3
MED	19.5	1.2	10.2	5.7	3.0	7.3	46.9
GYN	14.4	10.2	2.6	0.9	0.6	3.1	31.7
OB	4.2	6.6	0.5	0.9	0.5	7.5	20.3
PED	2.8	1.6	3.9	2.1	1.2	4.9	16.6
NEW	1.0	0.3	2.9	3.6	3.7	5.6	17.4
Total	**14.2**	**6.6**	**7.7**	**3.9**	**2.6**	**6.4**	**41.4**

FIGURE 1. Distribution of infections, by etiology, 1984



E. coli was the pathogen most frequently associated with urinary tract infections, followed by enterococci and *P. aeruginosa* (Table 7). *S. aureus* was the pathogen most often associated with surgical wound infections, followed by enterococci and *E. coli*. *P. aeruginosa* was the pathogen most frequently associated with lower respiratory tract infections, followed by *S. aureus* and *Klebsiella* spp. Coagulase-negative staphylococci were the pathogens most commonly associated with primary bacteremia, followed by *S. aureus* and *E. coli*.

When the pathogens causing infections at the five major sites were examined by service, interesting differences were noted (Table 8). On all six services, *E. coli* was the pathogen most often isolated from the urinary tract. Enterococci were the second most frequently isolated pathogens from the urinary tract on the obstetrics, gynecology, and medicine services, whereas *P. aeruginosa* was the second most commonly isolated pathogen from the urinary tract on the surgery and pediatrics services, and *Klebsiella* spp. were second on the newborn service. *S. aureus* was the pathogen most often associated with surgical wound infections on all services except gynecology, where *E. coli* was isolated most frequently. The pathogen most frequently associated with lower respiratory infections on all services was *P. aeruginosa*, with *S. aureus* second on all but the gynecology and newborn services. Coagulase-negative staphylococci were most often associated with primary bacteremia on the pediatrics, newborn, and surgery services, whereas *S. aureus* was the pathogen most frequently associated with bacteremia on the medicine and obstetrics services. *E. coli* and *Bacteroides* spp. were isolated with the highest frequency in association with primary bactere-

TABLE 6. The 15 most frequently isolated pathogens and their percentage distribution on each service, 1984

	Service							
Pathogen	**MED**	**SURG**	**OB**	**GYN**	**PED**	**NEW**	**Total isolates**	**%**
E. coli	19.6	16.2	21.2	29.8	11.4	9.3	5,266	17.8
P. aeruginosa	11.4	13.0	1.3	4.3	9.7	6.7	3,366	11.4
Enterococci	9.6	10.5	16.6	18.1	5.3	5.7	3,063	10.4
S. aureus	9.2	10.4	8.0	5.8	16.6	24.8	3,059	10.3
Klebsiella spp.	9.0	6.9	2.1	4.8	6.6	6.7	2,193	7.4
Coagulase-negative staphylococci	5.6	6.1	5.7	5.2	13.2	15.3	1,868	6.3
Enterobacter spp.	4.7	7.5	2.1	3.7	4.2	3.7	1,748	5.9
Candida spp.	7.0	4.9	1.1	2.2	7.6	3.8	1,620	5.5
Proteus spp.	5.6	5.4	3.4	5.3	0.3	1.0	1,522	5.1
Serratia spp.	2.1	2.9	0.2	0.3	1.4	1.3	691	2.3
Other fungi	2.3	1.5	0.1	0.1	1.2	1.0	496	1.7
Citrobacter spp.	1.5	1.5	1.1	0.8	1.0	0.8	414	1.4
Bacteroides spp.	0.6	1.4	4.6	2.8	0.3	0.2	355	1.2
Group B *Streptococcus*	0.8	0.5	7.9	3.8	1.2	6.2	348	1.2
Other anaerobes	0.9	0.9	4.8	2.0	0.3	0.2	300	1.0
All others*	10.1	10.4	19.8	11.0	19.7	13.3	3,253	11.1
Number of Isolates	11,304	14,596	1,024	1,016	590	1,032	29,562	100.0

*No other pathogen accounted for more than 3% of the isolates on any service.

mia on the gynecology service. *S. aureus* was most commonly associated with cutaneous infections and was followed by coagulase-negative staphylococci on all services except surgery and obstetrics. On surgery, *S. aureus* was first, followed by *P. aeruginosa*; on obstetrics, *E. coli* was isolated most frequently, followed by *S. aureus*.

Secondary Bacteremia. Secondary bacteremia was defined as a bloodstream infection with an organism that was also isolated from an infection at another site. Secondary bacteremia was reported most frequently by large teaching hospitals and least frequently by nonteaching hospitals (Table 9). Secondary bacteremia occurred most often on the pediatrics service in teaching hospitals, followed by the medicine, newborn, and surgery services, and it occurred least frequently on the obstetrics and gynecology services. In nonteaching hospitals, secondary bacteremia occurred most often on the medicine, obstetrics, and surgery services and least often on the newborn, gynecology, and pediatrics services. For all hospital categories, secondary bacteremia was associated less frequently with urinary tract, surgical wound, lower respiratory tract, and cutaneous infections than with infections, collectively, at "other" sites (Table 10). With respect to the four major sites, and excluding primary bacteremia, secondary bacteremia occurred most often following cutaneous infections. It occurred most frequently in all hospitals following infections with *Acinetobacter* spp., *Bacteroides* spp., *S. aureus*, *Serratia* spp., and coagulase-negative staphylococci (Table 11), but this varied greatly within each hospital category. For example, in nonteaching hospitals, *S. aureus* was the main pathogen that caused secondary bacteremia. In small teaching hospitals, the frequency of secondary bacteremia due to coagulase-negative staphylococci has nearly doubled

TABLE 7. The 15 most frequently isolated pathogens and their percentage distribution for each site of infection, 1984

	Site							
Pathogen	**UTI**	**SWI**	**LRI**	**BACT**	**CUT**	**Other**	**Total isolates**	**%**
E. coli	30.7	11.5	6.4	10.1	7.0	7.4	5,266	17.8
P. aeruginosa	12.7	8.9	16.9	7.6	9.2	6.7	3,366	11.4
Enterococci	14.7	12.1	1.5	7.1	8.8	7.0	3,063	10.4
S. aureus	1.6	18.6	12.9	12.3	28.9	14.6	3,059	10.3
Klebsiella spp.	8.0	5.2	11.6	7.8	3.8	4.6	2,193	7.4
Coagulase-negative staphylococci	3.4	8.3	1.5	14.9	11.5	11.6	1,868	6.3
Enterobacter spp.	4.8	7.0	9.4	6.3	4.5	3.9	1,748	5.9
Candida spp.	5.4	1.7	4.0	5.6	5.8	14.1	1,620	5.5
Proteus spp.	7.4	5.2	4.2	0.8	3.3	2.1	1,522	5.1
Serratia spp.	1.2	2.1	5.8	3.0	2.2	1.5	691	2.3
Other fungi	2.2	0.4	1.4	1.3	0.9	2.8	496	1.7
Citrobacter spp.	1.8	1.4	1.4	0.7	0.7	0.9	414	1.4
Bacteroides spp.	0.0	3.7	0.2	3.4	1.2	1.4	355	1.2
Group B *Streptococcus*	0.9	1.3	0.7	2.3	1.1	1.9	348	1.2
Other anaerobes	0.0	1.7	0.1	1.8	0.8	4.4	300	1.0
All others*	5.2	10.9	22.0	15.0	10.3	15.1	3,253	11.1
Number of Isolates	12,218	5,500	4,567	2,264	1,690	3,323	29,562	100.0

*No other pathogen accounted for more than 3% of the isolates at any site.

TABLE 8. Five most common pathogens isolated and percentage of total within each site and service, 1984

	Site									
	UTI		SWI		LRI		BACT		CUT	
Service	**Pathogen**	**%**	**Pathogen**	**%**	**Pathogen**	**%**	**Pathogen**	**%**	**Pathogen**	**%**
Medicine	*E. coli*	30.6	*S. aureus*	19.7	*P. aeruginosa*	16.6	*S. aureus*	14.4	*S. aureus*	26.3
	Enterococci	14.2	Enterococci	12.1	*S. aureus*	14.8	Coag-neg *staph.*	13.8	Coag-neg *staph.*	11.8
	P. aeruginosa	11.3	*P. aeruginosa*	9.3	*Klebsiella* spp.	12.2	*E. coli*	12.2	*P. aeruginosa*	11.0
	Klebsiella spp.	9.4	Coag-neg *staph.*	9.0	*Enterobacter* spp.	7.7	*P. aeruginosa*	9.2	Enterococci	9.1
	Proteus spp.	8.1	*E. coli*	6.9	*E. coli*	7.3	*Klebsiella* spp.	8.5	*Candida* spp.	6.6
Surgery	*E. coli*	29.2	*S. aureus*	19.0	*P. aeruginosa*	16.5	Coag-neg *staph.*	14.0	*S. aureus*	19.0
	P. aeruginosa	16.1	Enterococci	12.1	*S. aureus*	11.5	*S. aureus*	10.2	*P. aeruginosa*	12.9
	Enterococci	13.4	*E. coli*	11.5	*Enterobacter* spp.	11.4	*Enterobacter* spp.	9.2	Enterococci	10.7
	Proteus spp.	7.4	*P. aeruginosa*	9.7	*Klebsiella* spp.	11.2	Enterococci	9.1	Coag-neg *staph.*	10.1
	Klebsiella spp.	6.7	Coag-neg *staph.*	7.9	*Serratia* spp.	6.9	*Klebsiella* spp.	7.5	*E. coli*	7.9
Gynecology	*E. coli*	40.7	*E. coli*	15.1	*P. aeruginosa*	15.6	*E. coli*	16.0	*S. aureus*	18.8
	Enterococci	23.5	*S. aureus*	12.9	*Enterobacter* spp.	9.4	*Bacteroides* spp.	16.0	Coag-neg *staph.*	12.5
	Klebsiella spp.	6.7	Enterococci	12.9	*S. aureus*	6.3	Coag-neg *staph.*	8.0	*Morganella* spp.	12.5
	Proteus spp.	5.0	Coag-neg *staph.*	11.5	*Candida* spp.	6.3	*S. aureus*	8.0	Viruses	12.5
	P. aeruginosa	4.5	*Bacteroides* spp.	6.8	*Klebsiella* spp.	3.1	*Klebsiella* spp.	4.0	Other anaerobes	12.5
Obstetrics	*E. coli*	36.3	*S. aureus*	13.7	*P. aeruginosa*	7.7	*S. aureus*	14.3	*E. coli*	25.6
	Enterococci	28.8	*E. coli*	13.7	*S. aureus*	7.7	Other anaerobes	12.2	*S. aureus*	23.1
	Group B *strep.*	6.0	Enterococci	12.5	*Klebsiella* spp.	7.7	*E. coli*	8.2	Enterococci	10.3
	Coag-neg *staph.*	3.6	Coag-neg *staph.*	9.6	*E. coli*	7.7	Coag-neg *staph.*	6.1	Coag-neg *staph.*	7.7
	Proteus spp.	3.3	*Bacteroides* spp.	8.3	*Candida* spp.	7.7	Enterococci	6.1	Group B. *strep.*	5.1
Pediatrics	*E. coli*	30.4	*S. aureus*	34.8	*P. aeruginosa*	19.8	Coag-neg *staph.*	29.0	*S. aureus*	40.3
	P. aeruginosa	13.4	*E. coli*	10.6	*S. aureus*	11.6	*S. aureus*	14.0	Coag-neg *staph.*	19.4
	Enterococci	10.7	*P. aeruginosa*	10.6	*Klebsiella* spp.	9.3	*E. coli*	10.8	*E. coli*	10.4
	Klebsiella spp.	10.7	Coag-neg *staph.*	10.6	*Enterobacter* spp.	7.0	*Klebsiella* spp.	6.5	*Candida* spp.	7.5
	Candida spp.	10.7	Enterococci	7.6	*Candida* spp.	3.5	*Candida* spp.	6.5	Enterococci	4.5
Newborn	*E. coli*	35.2	*S. aureus*	23.1	*P. aeruginosa*	29.5	Coag-neg *staph.*	20.8	*S. aureus*	54.5
	Klebsiella spp.	15.5	Coag-neg *staph.*	23.1	*Klebsiella* spp.	15.2	Group B. *strep.*	15.3	Coag-neg *staph.*	12.6
	Coag-neg *staph.*	9.9	*P. aeruginosa*	15.4	Coag-neg *staph.*	9.8	Enterococci	9.9	*E. coli*	6.1
	Enterococci	8.5	Enterococci	7.7	*E. coli*	8.0	*E. coli*	9.4	Enterococci	4.8
	Candida spp.	8.5	*Klebsiella* spp.	7.7	*S. aureus*	7.1	*Klebsiella* spp.	8.4	*Enterobacter* spp.	4.2

since 1983 (*6*). In addition, *Bacteroides* spp., *S. aureus*, Group B *Streptococcus*, and *Acinetobacter* were frequently associated with secondary bacteremia in small teaching hospitals. In large teaching hospitals, no pathogen predominated as the causative agent of secondary bacteremia.

TABLE 9. Percentage of infections* with secondary bacteremia, by service and hospital category, 1984

	Service						
Hospital category	**SURG**	**MED**	**GYN**	**OB**	**NEW**	**PED**	**All services**
Nonteaching	3.6	4.3	1.6	4.0	2.9	0.0	3.8
Small teaching	5.0	5.5	1.8	3.1	5.2	6.8	4.9
Large teaching	6.5	8.5	1.8	2.5	6.5	8.6	6.8
All hospitals	**5.4**	**6.6**	**1.8**	**2.8**	**5.5**	**7.8**	**5.6**

*Excluding primary bacteremia

TABLE 10. Percentage of infection with secondary bacteremia, by site* and hospital category, 1984

	Site					
Hospital category	**UTI**	**SWI**	**LRI**	**CUT**	**Other†**	**All sites**
Nonteaching	3.1	3.5	3.3	4.1	8.9	3.8
Small teaching	2.7	4.2	6.1	9.4	9.6	4.9
Large teaching	3.9	6.0	5.9	9.4	14.3	6.8
All hospitals	**3.3**	**4.9**	**5.4**	**8.6**	**12.0**	**5.6**

*Excluding primary bacteremia.

†Most frequently associated with cardiovascular (70.8%) and intra-abdominal infections (10.5%).

TABLE 11. Ten pathogens with the highest percentage of associated secondary bacteremia, by hospital category, 1984

	Nonteaching		Small teaching		Large teaching		All hospitals	
Pathogen	**No. of infections**	**% with secondary bacteremia**	**No. of infections**	**% with secondary bacteremia**	**No. of infections**	**% with secondary bacteremia**	**No. of infections**	**% with secondary bacteremia**
Acinetobacter spp.	21	0.0	30	10.0	85	22.4	136	16.2
Bacteroides spp.	17	5.9	51	13.7	47	19.1	115	14.8
S. aureus	439	9.3	887	12.4	1,118	15.9	2,444	13.5
Serratia spp.	99	6.1	118	5.9	239	18.0	456	12.3
Coagulase-negative staphylococci	151	3.3	449	11.1	529	13.4	1,129	11.2
Group B *Streptococcus*	35	2.9	87	10.3	79	10.1	201	9.0
Klebsiella spp.	290	4.8	475	4.6	667	10.6	1,432	7.5
Other fungi	34	5.9	139	2.9	180	10.0	353	6.8
Morganella spp.	36	5.6	45	4.4	53	7.5	134	6.0
Other *Pseudomonas* spp.	63	0.0	66	3.0	39	12.4	218	6.0

Antimicrobial Resistance. Resistance was defined as the number of resistant isolates divided by the number of organisms that were either sensitive or resistant, multiplied by 100. Methicillin-resistant *S. aureus* was most commonly reported from the large teaching hospitals (Table 12). In fact, for all the antimicrobials listed in Table 12, resistance was most often reported from the large teaching hospitals.

The percentages of *E. coli*, *Klebsiella pneumoniae*, *Serratia marcescens*, and *P. aeruginosa* organisms that were resistant to aminoglycosides and selected beta-lactam antibiotics varied according to the three hospital categories (Tables 13-16). Aminoglycoside resistance was most common in *P. aeruginosa* and *S. marcescens*, and cefotaxime or moxalactam resistance was most common in *P. aeruginosa*.

TABLE 12. Antimicrobial resistance of *Staphyloccocus aureus*, 1984

	Number resistant (%)				
Hospital category	**Methicillin**	**Gentamicin**	**Clindamycin**	**Chloramphenicol**	**Erythromycin**
Nonteaching	23 (6.0)	38 (8.6)	41 (9.1)	19 (5.4)	58 (11.9)
Small teaching	43 (4.6)	50 (5.9)	65 (7.2)	25 (3.0)	107 (10.9)
Large teaching	140 (11.3)	106 (10.6)	140 (10.4)	84 (7.2)	245 (18.0)

TABLE 13. Antimicrobial resistance of *Escherichia coli*, 1984

	Number resistant (%)				
Hospital category	**Gentamicin**	**Tobramycin**	**Amikacin**	**Cefotaxime**	**Moxalactam**
Nonteaching	28 (2.4)	11 (1.4)	9 (1.6)	1 (1.2)	0 (0.0)
Small teaching	48 (2.6)	19 (1.5)	11 (2.2)	6 (3.2)	9 (7.1)
Large teaching	46 (2.0)	51 (2.6)	21 (1.7)	9 (1.3)	3 (1.0)

TABLE 14. Antimicrobial resistance of *Klebsiella pneumoniae*, 1984

	Number resistant (%)				
Hospital category	**Gentamicin**	**Tobramycin**	**Amikacin**	**Cefotaxime**	**Moxalactam**
Nonteaching	20 (4.7)	12 (3.7)	0 (0.0)	0 (0.0)	0 (0.0)
Small teaching	26 (3.6)	12 (2.3)	7 (2.7)	6 (8.6)	5 (8.2)
Large teaching	67 (6.7)	47 (5.9)	24 (4.2)	5 (1.3)	7 (3.1)

TABLE 15. Antimicrobial resistance of *Serratia marcescens*, 1984

	Number resistant (%)				
Hospital category	**Gentamicin**	**Tobramycin**	**Amikacin**	**Cefotaxime**	**Moxalactam**
Nonteaching	10 (6.1)	11 (8.3)	6 (5.4)	3 (7.3)	0 (0.0)
Small teaching	13 (7.2)	16 (11.0)	3 (3.7)	4 (6.1)	6 (10.3)
Large teaching	41 (11.6)	52 (18.4)	18 (8.7)	21 (11.3)	8 (7.2)

Mortality. Data from individual hospitals are included in the mortality analyses if the hospital assessed and reported the relationship of infection to death for more than 50% of the infections in patients who died while hospitalized. The 42 hospitals that met this criterion reported a total of 22,432 infections; among the 1,253 patients who died, there were 1,811 infections for which the relationship of the infection to death was recorded. Approximately 1% of all nosocomial infections caused death, and 3% contributed to death (Table 17). Infections were more often reported to cause or contribute to death in small teaching and in nonteaching hospitals than in large teaching hospitals. Among infected patients who died while hospitalized, 9% of the infections reportedly caused death, 38% contributed to it, and 37% were not related to death; in 15% of these infections, the relationship of the infection to death could not be determined (Table 18).

TABLE 16. Antimicrobial resistance of *Pseudomonas aeruginosa*, 1984

	Number resistant (%)				
Hospital category	Gentamicin	Tobramycin	Amikacin	Cefotaxime	Moxalactam
Nonteaching	111 (15.1)	43 (6.8)	23 (5.1)	74 (64.3)	30 (30.6)
Small teaching	91 (9.0)	40 (5.0)	33 (6.7)	163 (59.1)	137 (59.1)
Large teaching	228 (15.7)	117 (7.9)	54 (5.6)	180 (36.1)	121 (28.4)

TABLE 17. Percentage of infections reported as having caused or contributed to death of the patient, 1984

Hospital category	Number of infections	Percentage that caused death	Percentage that contributed to death
Nonteaching	3,553	0.7	3.9
Small teaching	8,609	1.1	3.1
Large teaching	10,270	0.5	2.8
Total	**22,432**	**0.7**	**3.1**

TABLE 18. Relationship of infection to death by hospital category, 1984*

	Number (%)				
Hospital category	Caused death	Contributed to death	Not related to death	Unknown	Total
Nonteaching	23 (6.7)	139 (40.6)	138 (40.4)	42 (12.3)	342 (100)
Small teaching	96 (12.5)	268 (35.0)	251 (32.8)	151 (19.7)	766 (100)
Large teaching	47 (6.7)	287 (40.8)	288 (41.0)	81 (11.5)	703 (100)
Total	**166 (9.2)**	**694 (38.3)**	**677 (37.4)**	**274 (15.1)**	**1,811 (100)**

*There were 1,811 infections in 1,253 patients who died.

Discussion

Nosocomial infections remain an important cause of morbidity and mortality in U.S. hospitals. Data from NNIS, the only national source of prospectively collected data on hospital-acquired infections, show that the overall rate of nosocomial infections during 1984 was 3.4 infections per 100 patients discharged. This is similar to the infection rate reported for the 3-year period 1980-1982 (*4*) and for 1983 (*6*). By comparison, the Study on the Efficacy of Nosocomial Infection Control (SENIC) found that a nosocomial infection develops in 5%-6% of hospitalized patients (*8*). SENIC was a retrospective study involving a representative sample of U.S. hospitals in 1975-1976. NNIS data suggest that the true incidence of nosocomial infections is underestimated. Factors contributing to the underestimation include variability of the intensity of surveillance and availability of laboratory support, especially in diagnostic virology. Since identification of nosocomial viral infections depends on both laboratory detection and surveillance intensity, hospitals without virology laboratory support will be unlikely to detect most of these infections.

Since 1980 (*4*), nosocomial infection rates have been consistently highest in large teaching hospitals and lowest in nonteaching hospitals for all services and sites of infection, suggesting that the three-level stratification effectively defines hospital categories in which patients have different levels of risk for acquiring nosocomial infections. This difference in risk undoubtedly reflects severity of underlying illness (patient mix) and the extent to which invasive diagnostic and therapeutic procedures are performed in these hospitals.

As in 1980-1982 (*4*) and in 1983 (*6*), the infection rates were highest on the surgery and medicine services, probably because of their high-risk patient populations. The lowest infection rates were on the pediatrics and newborn services. One explanation for this lower rate may be that in NNIS hospitals, there are fewer high-risk children and newborns than adults, particularly in the small hospitals. Furthermore, most of the infants included in the newborn service are in well-baby nurseries, where the infection risk is expected to be lower. Another factor that may help explain the lower rates of infection on the pediatrics and newborn services is that only a small proportion of NNIS hospitals have diagnostic virology laboratories; therefore, many viral infections probably go undetected. Since children more often acquire nosocomial viral infections than adults (*9*), and since in one study viruses accounted for approximately 14% of nosocomial infections in children (*10*), NNIS hospitals are probably underreporting viral infections. In addition, other factors, such as the short time that many pediatric patients are hospitalized and the frequent use of isolation precautions on the pediatrics and newborn services, may reduce the incidence of nosocomial infections on these services.

In 1984, infection rates on different services and at different sites of infection within the three hospital categories varied little from those reported for 1983 (*6*). Since 1980-1982 (*4*), the primary bacteremia and lower respiratory tract infection rates have increased. The overall lower respiratory tract infection rate surpassed the rate of surgical wound infections in 1984. Whether this is an artifact of reporting or a true shift in the rates is not known.

Specimens for microbiologic testing were obtained from 90% of the patients reported to have a nosocomial infection. Aerobic bacteria were the most commonly identified etiologic agents. Anaerobic bacteria, fungi, parasites, and viruses were seldom reported, reflecting in part the frequency with which these pathogens are looked for, as well as the diagnostic laboratory capabilities of the hospitals.

E. coli was the most frequently identified pathogen on the four adult services, reflecting the fact that this organism was the primary cause of urinary tract infections on these services.

In contrast, *S. aureus* was the pathogen most often identified on the newborn and pediatrics services. Coagulase-negative staphylococci were the second most frequent cause of nosocomial infections on the newborn and pediatrics services and were an important cause of bacteremia on all services except gynecology and obstetrics. Recent studies suggest that the increasing use of long-line catheters may be contributing to the emergence of coagulase-negative staphylococci as an important cause of primary bacteremia (*11,12*).

Previous analyses of NNIS data have suggested that secondary bacteremia carries an increased risk of death (*13*). In all hospitals, the major sites of infection that were most likely to result in secondary bacteremia were cutaneous infections, followed by surgical wound and lower respiratory tract infections. Infections at sites other than the four major sites were, collectively, more frequently associated with secondary bacteremia. These include cardiovascular and intra-abdominal infections. An increase in cardiovascular surgery and in the use of long-line venous and arterial catheters may have accounted for the rise since 1983 in the percentage of infections associated with the cardiovascular system (*6*). Because of the increased risk of death associated with secondary bacteremia, these infections continue to be a high priority for prevention and control (*13*).

As in the past, the incidence of methicillin-resistant *S. aureus* (MRSA) infections was highest at large teaching hospitals (*4,6,14*), and between 1983 and 1984, these infections increased by more than 25% in each hospital category (*6*). Since 1983, the proportion of *S. aureus* organisms resistant to gentamicin and clindamycin increased at small teaching and at nonteaching hospitals, but at large teaching hospitals the proportion resistant to gentamicin decreased and that resistant to clindamycin remained about the same (*6*). The factors responsible for these resistance trends require further study. Recent work suggests that risk factors for MRSA may differ bv type of hospital (*15*).

In 1984, compared with 1983, the proportion of *K. pneumoniae* organisms resistant to gentamicin and tobramycin decreased in large teaching hospitals and increased in nonteaching hospitals; however, resistance to amikacin increased in large teaching hospitals and decreased in both nonteaching and small teaching hospitals (*6*). Since 1982, the resistance of *P. aeruginosa* to both cefotaxime and moxalactam has increased in the small teaching hospitals, but the trend has been variable in nonteaching hospitals (*4,6*). Over the same period cefotaxime resistance has continued to decrease, and moxalactam resistance has been rising in the large teaching hospitals (*4,6*). Since the proportion of isolates tested against cefotaxime and moxalactam was small, these data should be interpreted with caution.

When compared with NNIS mortality data for 1980-1982 (*4*) and 1983 (*6*), the overall percentage of infections reported to cause or contribute to death in 1984 has not changed significantly. Since 1980, the large teaching hospitals have reported a slightly lower percentage of infections each year as causing or contributing to a patient's death (*4,6*). The small teaching hospitals reported about the same frequency, and the nonteaching hospitals reported a slight increase each year (*4,6*). Mortality data should be interpreted with caution, since standard criteria for assessing the relationship of infection to death do not exist.

This nationwide nosocomial surveillance system is expanding in four directions (*6*). First, microcomputer software called the Interactive Data Entry and Analysis System (IDEAS) has been developed to support nosocomial infection surveillance activities of NNIS hospitals. Beginning in October 1984, IDEAS was pilot tested in three hospitals and is now being used in 22 additional hospitals. This information management system not only helps to improve the quality and timeliness of nosocomial infection data collected in NNIS, but it also assists infection control practitioners in conducting more effective and efficient surveillance in their institutions. Second, additional hospitals are being added to the surveillance system so that the data obtained will be from a more representative sample of all acute-care hospitals in the United States. Since recruitment began in March 1985, 10 hospitals have been added and additional hospitals are being considered for enrollment. Third, in July 1985, the feasibility of collecting data on antimicrobial usage in NNIS hospitals was assessed. Hospitals with computerized pharmacy records wishing to participate in the study will report on the use of antimicrobial agents so that for selected nosocomial bacterial pathogens the relationship between usage and resistance can be evaluated. Fourth, strategies are being developed for determining a more sensitive indicator of patients' risk based on characteristics of both the patient and the hospital (such as the three size categories used in this report). When various levels of nosocomial infection risks can be calculated, infection rates among hospitals can be compared and the distribution of risks can be standardized; in addition, hospital-specific infection rates and secular trends can be evaluated more effectively.

References

1. Haley RW, Schaberg DR, Crossley KB, Von Allmen SD, McGowan JE Jr. Extra charges and prolongation of stay attributable to nosocomial infections: a prospective interhospital comparison. Am J Med 1981;70:51-8.
2. Haley RW, Schaberg DR, Von Allmen SD, McGowan JE. Estimating the extra charges and prolongation of hospitalization due to nosocomial infections: a comparison of methods. J Infect Dis 1980;141:248-57.
3. McGowan JE. Cost and benefit: a critical issue for hospital infection control. Am J Infect Control 1982;10:100-8.
4. CDC. Nosocomial infection surveillance, 1980-1982. In: Surveillance Summaries (published four times a year). 1983;32(No.4SS):1SS-16SS.
5. Hughes JM, Jarvis WR. Epidemiology of nosocomial infections. In: Lennette EH, Balows A, Hausler WJ Jr, Shadomy HJ, eds. Manual of clinical microbiology. Washington, D.C.: American Society for Microbiology 1985:99-104.
6. CDC. Nosocomial infection surveillance, 1983. In: Surveillance Summaries (published four times a year). 1984;33(no. 2SS):9SS-21SS.
7. American Hospital Association. American Hospital Association guide to the health care field. Chicago: American Hospital Association, 1983.
8. Haley RW, Hooton TM, Culver DH, et al. Nosocomial infections in U.S. hospitals, 1975-1976: estimated frequency by selected characteristics of patients. Am J Med 1981;70:947-59.
9. Valenti WM, Hall CB, Douglas RG Jr, Menegus MA, Pincus PH. Nosocomial viral infections: 1. Epidemiology and significance. Infect Control 1980;1:33-7.
10. Welliver RC, McLaughlin S. Unique epidemiology of nosocomial infection in a children's hospital. Am J Dis Child 1984;138:131-5.
11. Munson DP, Thompson TR, Johnson DE, Rhame FS, Van Drunen N, Ferrieri P. Coagulase-negative staphylococcal septicemia: experience in a newborn intensive care unit. J Pediatr 1982;101:602-5.
12. Christensen GD, Bisno AL, Parisi JT, McLaughlin B, Hester MG, Luther RW. Nosocomial septicemia due to multiply antibiotic-resistant *Staphylococcus epidermidis*. Ann Intern Med 1982;96:1-10.
13. Hughes JM, Munn VP, Jarvis WR, Culver DH, Haley RW. Mortality associated with nosocomial infections in the United States, 1975-1981. Abstract 701. Presented at the 22nd Interscience Conference on Antimicrobial Agents and Chemotherapy, Miami Beach, Florida, 1982 October 4-6.
14. Haley RW, Hightower AW, Khabbaz RF, et al. The emergence of methicillin-resistant *Staphylococcus aureus* infections in United States hospitals. Ann Intern Med 1982; 97:297-308.
15. Jarvis WR, Thornsberry C, Boyce J, Hughes JM. Methicillin-resistant *Staphylococcus aureus* at children's hospitals in the United States. Pediatr Infect Dis 1985; 4:651-5.

Copies can be purchased from:
Superintendent of Documents
U.S. Government Printing Office
Washington, D.C. 20402
Telephone: (202) 783-3238

State and Territorial Epidemiologists and State Laboratory Directors

The contributions of the State and Territorial Epidemiologists and the State Laboratory Directors to this report are gratefully acknowledged. The persons listed were in the positions shown as of March 1, 1986.

State	Epidemiologists	Laboratory Directors
Alabama	Wallace E. Birch, DVM	James L. Holston, Jr., DrPH
Alaska	John P. Middaugh, MD	Harry J. Colvin, PhD
Arizona	Glyn G. Caldwell, MD	Jon M. Counts, DrPH
Arkansas	Thomas C. McChesney, DVM	Robert L. Horn
California	James Chin, MD	G. W. Fuhs, DrSc
Colorado	Stanley W. Ferguson, PhD	Ronald L. Cada, DrPH
Connecticut	James L. Hadler, MD, MPH	Jesse Tucker, PhD
Delaware	Paul R. Silverman, DrPH	Mahadeo P. Verma, PhD
District of Columbia	Martin E. Levy, MD	James B. Thomas, DSc
Florida	E. Charlton Prather, MD	Eldert C. Hartwig, ScD
Georgia	R. Keith Sikes, DVM	Frank M. Rumph, MD
Hawaii	Arthur P. Liang, MD, MPH	Robert Katasse, MD
Idaho	Charles D. Brokopp, DrPH	D. W. Brock, DrPH
Illinois	Byron J. Francis, MD	Harry C. Bostick
Indiana	Charles L. Barrett, MD	T. L. Eddleman
Iowa	Laverne A. Wintermeyer, MD	W. J. Hausler, Jr., PhD
Kansas	Robert French, Acting	Roger H. Carlson, PhD
Kentucky	M. Ward Hinds, MD	B. F. Brown, MD
Louisiana	Joyce B. Mathison, MD	Henry Bradford, PhD
Maine	Kathleen F. Gensheimer, MD	Philip W. Haines, DrPH
Maryland	Ebenezer Israel, MD	J. Mehsen Joseph, PhD
Massachusetts	George F. Grady, MD	George F. Grady, MD
Michigan	Kenneth R. Wilcox, Jr., MD	George R. Anderson, DVM
Minnesota	Michael Osterholm, PhD, MPH	Harold Markowitz, PhD, MD
Mississippi	Fred E. Thompson, MD	R. H. Andrews, MPH
Missouri	H. Denny Donnell, Jr., MD	Elmer R. Spurrier, DrPH
Montana	Judith K. Gedrose, RN	Douglas Abbott, PhD
Nebraska	Paul A. Stoesz, MD	John Blosser
Nevada	George E. Reynolds, MD, Acting	George Reynolds, MD
New Hampshire	Eugene Schwartz, MD	Veronica C. Malmberg
New Jersey	William E. Parkin, DVM	Bernard F. Taylor, PhD
New Mexico	Harry F. Hull, MD	Loris W. Hughes, PhD
New York State	Dale Morse, MD	David O. Carpenter, MD
New York City	Stephen Schultz, MD	Bernard Davidow, PhD, Acting
North Carolina	J.N. MacCormack, MD	Mildred A. Kerbaugh
North Dakota	James L. Pearson, DrPH	Jane S. Robb, Acting
Ohio	Thomas J. Halpin, MD, MPH	Gary D. Davidson, DrPH
Oklahoma	Gregory R. Istre, MD	Garry L. McKee, PhD
Oregon	Laurence Foster, MD, MPH	Michael R. Skeels, PhD
Pennsylvania	Ernest J. Witte, VMD	Vern Pidcoe, DrPH
Rhode Island	Richard A. Keenlyside, MBBS	Raymond G. Lundgren, Jr., PhD
South Carolina	Richard L. Parker, DVM	Arthur F. DiSalvo, MD
South Dakota	Kenneth A. Senger	A. Richard Melton, DrPH
Tennessee	Robert H. Hutcheson, Jr., MD	Michael W. Kimberly, DrPH
Texas	Charles E. Alexander, MD	Charles E. Sweet, DrPH
Utah	Craig R. Nichols, MPA	Francis M. Urry, PhD
Vermont	Richard L. Vogt, MD	Katherine A. Kelley, DrPH
Virginia	Grayson B. Miller, Jr., MD	Frank W. Lambert, Jr., DrPH
Washington	John M. Kobayashi, MD	Jack Allard, PhD
West Virginia	Loretta E. Haddy, MS	John W. Brough, DrPH
Wisconsin	Jeffrey P. Davis, MD	Ronald H. Laessig, PhD
Wyoming	Harry C. Crawford, MD	Donald T. Lee, DrPH
Guam	Robert L. Haddock, DVM	Luis P. Flores
Ponape*	Eliuel K. Pretrick, MO	Vacant
Marshall Islands	Isao Kisino	Vacant
Northern Mariana Is.*	Jose T. Villagomez, MO	Vacant
American Samoa	Julia L. Lyons, MD, MPH	Vacant
Palau*	Anthony H. Polloi, MO, Acting	Vacant
Puerto Rico	Jose G. Rigau, MD	Jose L. Villamil, PhD
Virgin Islands	John N. Lewis, MD	Norbert Mantor, PhD

---

*Formerly Trust Territory of the Pacific Islands.

☆U.S. Government Printing Office: 1986-746-149/40002 Region IV

UNITED STATES GOVERNMENT PRINTING OFFICE
SUPERINTENDENT OF DOCUMENTS
Washington, D.C. 20402

OFFICIAL BUSINESS
Penalty for Private Use, $300

BULK RATE
POSTAGE & FEES PAID
GPO
Permit No. G-26

MMWR NEWAROOON DR 376 F   1 **
NEWARK PUBLIC LIBR   **
5 WASHINGTON ST   **
NEWARK   NJ 07101   **
                              **

HHS Publication No. (CDC) 86-8017

Redistribution using indicia is illegal.

35/2S

CENTERS FOR DISEASE CONTROL

December 19, 1986/Vol. 35/No. 2S

MMWR

MORBIDITY AND MORTALITY WEEKLY REPORT

Supplement

PREMATURE MORTALITY IN THE UNITED STATES

Public Health Issues in the Use of Years of Potential Life Lost

U.S. DEPARTMENT OF HEALTH AND HUMAN SERVICES
PUBLIC HEALTH SERVICE
CENTERS FOR DISEASE CONTROL
EPIDEMIOLOGY PROGRAM OFFICE
DIVISION OF SURVEILLANCE AND EPIDEMIOLOGIC STUDIES
ATLANTA, GEORGIA 30333

HE 20.7009:

READEX FICHE best copy available

Supplements to the *MMWR* are published by the Epidemiology Program Office, Centers for Disease Control, Public Health Service, U.S. Department of Health and Human Services, Atlanta, Georgia 30333.

SUGGESTED CITATION

Centers for Disease Control. Premature mortality in the United States: public health issues in the use of years of potential life lost. *MMWR* 1986;35(suppl no. 2S):[inclusive page numbers].

Premature Mortality in the United States: Public Health Issues in the Use of Years of Potential Life Lost

INTRODUCTION

Mortality statistics are frequently used to quantitate the extent of public health problems and to determine the relative importance of the various causes of death. Analyses of crude and age-adjusted death rates have traditionally played an important role in this process; they enable priorities to be set and progress towards the achievement of public health goals to be monitored. Although these rates are important measures of the nation's health status, they often fail to tell the entire story of temporal changes in mortality. Since most deaths occur among persons in older age groups, crude and age-adjusted mortality data are dominated by the underlying disease processes of the elderly.

Alternative measures have been proposed to reflect the mortality trends of younger age groups. These measures provide a more accurate picture of premature mortality by weighting deaths occurring at younger ages more heavily than those occurring in older populations. One of these measures is the years of potential life lost (YPLL).

HISTORICAL PERSPECTIVES ON PREMATURE MORTALITY

In 1947, as a supplement to crude mortality figures, Dempsey introduced a method of quantifying premature mortality to measure the changing mortality consequences of tuberculosis (*1*). She tabulated the "potential years of life lost" by subtracting the age at death from the life expectancy at birth for persons who died during 1942 from heart disease, cancer, and tuberculosis. Greville subsequently altered this calculation, weighting each death by the life expectancy remaining at death (*2*). He also introduced the use of life tables calculated on the assumption that the given disease had been eliminated as a cause of death for all older age groups. This adjustment (the "zero-mortality assumption") only slightly changed the overall distribution of years of life lost and did not affect the rank order of causes (*3*).

Haenszel proposed directly age adjusting death rates to a standard population and then computing years of life lost from the resulting standardized deaths (*4*). Studies comparing temporal trends of premature mortality in populations widely separated in time have used a similar approach (*5,6*). In addition, standardized rates of "potentially productive years of life lost" have been developed to adjust for different age structures among populations and to describe race- and sex-specific patterns of premature mortality (*7*).

The use of premature mortality measures has recently become somewhat more common as a mechanism for establishing public health priorities (*7-10*). For this reason, the CDC introduced Table V of the *MMWR* in 1982. This table appears monthly and ranks the leading causes of premature mortality (*11*).

METHODOLOGIC ALTERNATIVES IN YPLL CALCULATION

Although many authors have emphasized the importance of the concept of premature mortality, there is no consensus on a functional definition or the best means of deriving a quantitative measure. Such a measure is necessary to monitor yearly trends in the pattern of premature mortality as well as to compare the relative importance of specified causes of death. This report will focus on YPLL, the measure of premature mortality used at CDC for the preparation of Table V of the *MMWR*. This section documents the current method of calculating YPLL used at CDC and discusses several adaptations of this measure for use in different situations.

Present Method of Calculation

For routine publication of Table V, YPLL is defined as the number of years of potential life lost by each death occurring before a predetermined end point, set at age 65 years. CDC calculates YPLL over the age range from birth to 65 years using age-specific death rates for 15 selected causes and supplementary data on causes of infant mortality, provided yearly by the National Center for Health Statistics (NCHS) (*12*). For timeliness, these data are estimated from a 10% sample of all death certificates filed during the year. Causes of death are grouped by an algorithm devised by NCHS to reflect the underlying cause of death as reported on the death certificate and the pathophysiologic process or organ system involved (i.e., malignant neoplasms or diseases of the heart).

The cause- and age-specific death rate is multiplied by the estimated population in that age range (provided by the Bureau of the Census) to determine the number and age distribution of deaths attributable to a specific cause (*13*). The number of deaths for each age is then multiplied by the years of life lost (the difference between the designated 65-year end point and the midpoint of the age range) to give an age-specific YPLL. For example, in the population 15-24 years old, the midpoint is 20 years and the YPLL is 45 years. Finally, the age-specific YPLLs are summed to give a total YPLL for each cause. The resulting distribution of YPLL, by cause, appears monthly as Table V (Table 1).

TABLE 1. Estimated years of potential life lost (YPLL) before age 65 and mortality, by cause of death, United States, 1984

Cause of mortality (Ninth Revision ICD)	YPLL (in thousands) for persons dying in 1984	Cause-specific mortality (rate/100,000)
ALL CAUSES (Total)	11,761	866.7
Unintentional injuries (E800-E949)	2,308	40.1
Malignant neoplasms (140-208)	1,803	191.6
Diseases of the heart (390-398, 402, 404-429)	1,563	324.4
Suicide and homicide (E950-E978)	1,247	20.6
Congenital anomalies (740-759)	684	5.6
Prematurity (765, 769)	470	3.0
Sudden infant death syndrome (798)	314	2.0
Cerebrovascular diseases (430-438)	266	65.6
Chronic liver diseases and cirrhosis (571)	233	11.3
Pneumonia and influenza (480-487)	163	25.0
Chronic obstructive pulmonary diseases (490-496)	123	29.8
Diabetes mellitus (250)	119	15.6

A persistent controversy in the calculation of YPLL concerns the definition of an upper end point. The CDC method of calculation does not count deaths of persons 65 years or older and assumes only 5 years of life lost for deaths of persons aged 55-64 years. In 1984, however, the remaining life expectancy for a 60-year-old person was 20.4 years (*12*). Thus, deaths in older age groups are underrepresented by the upper age limit of 65 years. However, this method preserves the emphasis on causes of mortality among younger persons.

Life-Expectancy Method

An alternative form of YPLL can be calculated that emphasizes the total life expectancy remaining at the time of death (Table 2). Age-specific death rates and population estimates are obtained from the same sources, but would not be truncated at age 65 (*12,13*). The number of deaths is estimated as above, but the YPLL for each death is calculated by taking the difference between the midpoint of the age range and the remaining life expectancy at that age from an abridged life table published yearly by NCHS (*12*). This table assumes that the risk of death in future years is approximated by the age-specific death rates for all causes in effect for the past year. For example, in the 75- to 84-year age group, the remaining life expectancy at the midpoint (80 years) was 8.2 years in 1984.

TABLE 2. Estimated years of potential life lost (YPLL)* and mortality, by cause of death, United States, 1984

Cause of mortality (Ninth Revision ICD)	YPLL (in thousands) for persons dying in 1984	Cause-specific mortality (rate/100,000)
ALL CAUSES (Total)	33,581	866.7
Diseases of the heart (390-398, 402, 404-429)	9,400	324.4
Malignant neoplasms (140-208)	7,171	191.6
Unintentional injuries (E800-E949)	3,381	40.1
Suicide and homicide (E950-E978)	1,833	20.6
Cerebrovascular diseases (740-759)	1,735	65.6
Chronic obstructive pulmonary diseases (490-496)	903	29.8
Congenital anomalies (740-759)	820	5.6
Pneumonia and influenza (480-487)	691	25.4
Chronic liver diseases and cirrhosis (571)	585	11.3
Prematurity (765, 769)	548	3.0
Diabetes mellitus (250)	525	15.6
Sudden infant death syndrome (798)	361	2.0

*Calculated by the remaining-life-expectancy method.

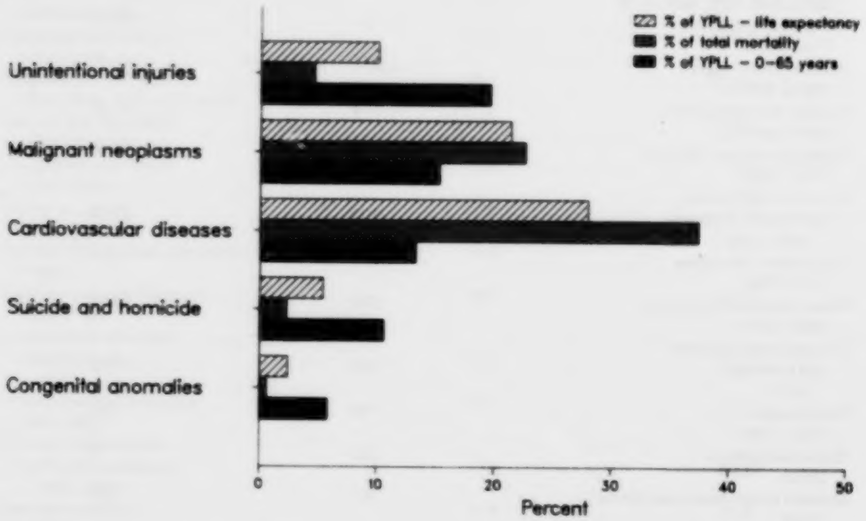
The number of deaths in each age group, grouped by underlying cause of death, is then multiplied by the remaining life expectancy at the midpoint of each age range, and the resulting numbers for all age groups are summed to obtain the total YPLL for this cause. Not surprisingly, this substantially changes both the total YPLL and the relative rankings for each cause (Table 3). When derived by the remaining-life-expectancy method, the proportion of YPLL attributable to each individual cause more closely resembles the pattern of cause-specific mortality than that of YPLL before age 65 (Figure 1).

TABLE 3. Rankings of the 10 leading causes of mortality and years of potential life lost (YPLL), by method of calculation, United States, 1984

Ranking	Crude mortality	YPLL—0-65 years	YPLL—life expectancy
1	Heart disease	Unintentional injuries	Heart disease
2	Malignant neoplasms	Malignant neoplasms	Malignant neoplasms
3	Cerebrovascular disease	Heart disease	Unintentional injuries
4	Unintentional injuries	Suicide and homicide	Suicide and homicide
5	COPD*	Congenital anomalies	Cerebrovascular disease
6	Pneumonia/influenza	Prematurity	COPD*
7	Suicide and homicide	Sudden infant death syndrome	Congenital anomalies
8	Diabetes	Cerebrovascular disease	Pneumonia/influenza
9	Cirrhosis	Cirrhosis	Cirrhosis
10	Congenital anomalies	Pneumonia/influenza	Diabetes

*Chronic obstructive pulmonary disease and allied conditions.

FIGURE 1. Percentage of years of potential life lost (YPLL)* and total mortality, by cause of death, United States, 1984



*Calculated by *MMWR* method (0-65 years) and by remaining-life-expectancy method

Mortality Groups

Another issue in the calculation of YPLL concerns the grouping of underlying causes of death as provided by NCHS. As a measure of mortality, YPLL underestimates the importance of diseases that contribute to, but are not recorded as, the underlying cause of death. A complete listing of any condition contributing to death is available from the NCHS tape "Mortality—Multiple Causes of Death," on which each condition recorded on the death certificate as either a contributing or an underlying cause of death is coded according to the *International Classification of Diseases*, Ninth Revision, Clinical Modification (ICD-9 CM).

In addition, it is impossible at present to calculate YPLL on a timely basis for those diseases or disease categories that are not reported separately by NCHS, such as a composite category of YPLL attributable to all infectious diseases. Age-specific death rates are supplied annually by NCHS for only the 15 leading causes of mortality and certain causes of infant mortality. For example, no summary data on mortality due to infectious diseases would be available in the annual report other than the categories of pneumonia and influenza or septicemia (*12*). However, the tape "Mortality—Underlying Cause of Death" contains detailed listings of all deaths, with the coded underlying cause of death. This tape is available from NCHS, but only after a lag time of several years after publication of data from the 10% sample. Therefore, although these data allow for a more individualized design of mortality groups by ICD-9 CM codes, they are not useful when timeliness is important.

Age Limits

Some research may benefit from alterations in the YPLL age limits, either to emphasize the effects of a condition in a population at increased risk of illness or to study a particular subpopulation of interest. For example, a recent review of the premature mortality consequences of the acquired immunodeficiency syndrome (AIDS) was limited to persons aged 25-44, the ages with the highest incidence of illness (*14*). The most common use of this approach in the literature is to study the working years of life lost, calculated over the age range from age 15 or 20 to 65 years (*3,4,6,7*).

YPLL Rates and Age Adjustment

When yearly trends in YPLL are compared for dynamic populations or for substantially different populations, it may be helpful to calculate YPLL rates per 1,000 persons in the age range. Use of the YPLL rate adjusts the total and cause-specific YPLL for differences in total population. For example, although the total YPLL increased 0.4% from 1983 to 1984, the rate of YPLL per 1,000 persons decreased by 0.4% (*15*). Similarly, if two population groups have different age structures, it is preferable to compare standardized or age-adjusted YPLL rates. This rate is derived by multiplying the age-specific death rates by a reference population, either directly adjusting one group to the other or adjusting both groups to an external standard. As an example, when one compares rates of YPLL for whites with those for American Indians and Alaskan Natives, it may be more accurate to adjust the rates for age because of the marked difference in the age structure of the comparison groups (*16*).

TRENDS IN YPLL AND YPLL RATES, 1979-1984

Premature mortality in the United States, as measured by YPLL, decreased in the 6-year period 1979-1984 (Figure 2). The total years of potential life lost before age 65 from all causes decreased from 13.1 million YPLL in 1979 to 11.8 million in 1984, a difference of 10.4%. Over 40% of this change in the total YPLL is due to a decrease in premature mortality attributable to unintentional injuries.

YPLL due to 10 of the 12 leading causes decreased during this time frame (Table 4). The largest proportionate decreases were for chronic liver disease and cirrhosis (down 22.3%), unintentional injuries (down 19.2%), and pneumonia and influenza (down 14.7%). In addition, a 13.1% decrease occurred in infant deaths associated with premature birth, that is, deaths coded with the ICD-9 rubric 765 or 769—respiratory distress syndrome and disorders associated with short gestation and unspecified low birth weight. Only YPLL from chronic obstructive pulmonary disease and allied conditions (up 20.6%) and diabetes mellitus (up 7.2%) increased.

FIGURE 2. Years of potential life lost (YPLL) before age 65, from all causes, United States, 1979-1984

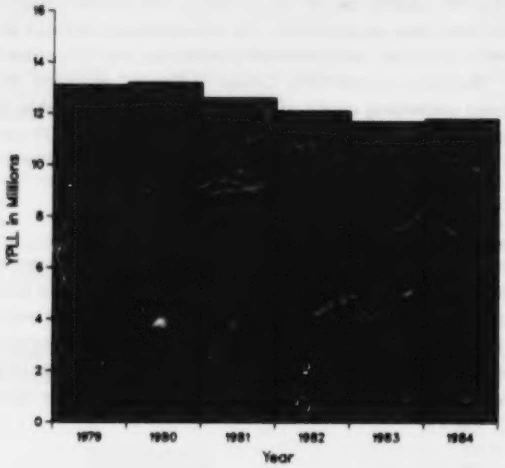


TABLE 4. Years of potential life lost (YPLL) before age 65 and percent change, selected causes, United States, 1979 and 1984

Cause of Mortality	YPLL (in thousands) 1979	YPLL (in thousands) 1984	Percent change 1979-1984
ALL CAUSES	13,123	11,761	-10.4
Unintentional injuries	2,858	2,308	-19.2
Heart disease	1,678	1,563	-6.9
Malignant neoplasms	1,829	1,803	-1.4
Suicide and homicide	1,357	1,247	-8.1
Prematurity	541	470	-13.1
Congenital malformations	749	684	-8.7
Sudden infant death syndrome	320	314	-1.9
Cerebrovascular disease	290	266	-8.3
Chronic liver disease	300	233	-22.3
Pneumonia and influenza	191	163	-14.7
Diabetes mellitus	111	119	+7.2
Chronic obstructive pulmonary disease	102	123	+20.6

The rate of YPLL per 1,000 persons less than 65 years of age for all causes of death decreased by 12.8% from 1979 to 1984. The population in the age range increased 5.5 million persons, augmenting the decrease in the total YPLL. YPLL rates also decreased for these same 10 leading causes of premature mortality (Figure 3).

VARIATIONS IN THE PATTERN OF YPLL

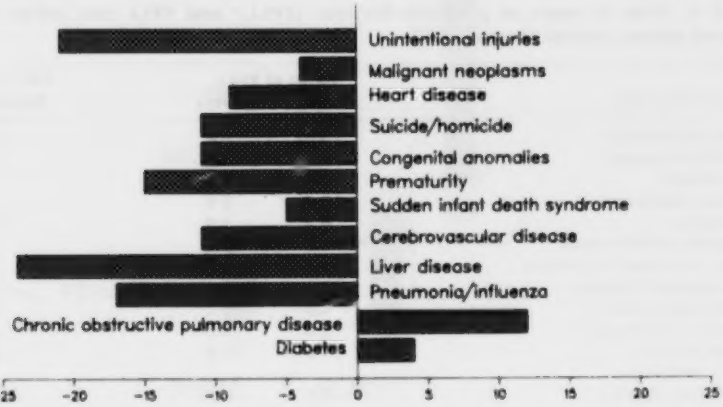
In addition to the programmatic implications of year-to-year changes in the total and cause-specific rates of YPLL, there may be marked differences in the rankings of the causes of YPLL among population subgroups. These differences can indicate populations with an increased risk of premature mortality (e.g., males compared with females) or identify individual causes of mortality of particular importance in certain of these groups, such as homicide among young black males. Interventions designed to alter this risk could then be targeted towards groups at highest risk, thereby increasing the effectiveness of those interventions. In this section, we examine differences in YPLL by race and sex and discuss its potential uses at the state and local levels.

Racial Variations in YPLL

Rates of premature mortality differ considerably among racial groups in the United States. During 1982, blacks had a YPLL rate from all causes of 99.2/1,000 population, a rate nearly twice that of whites (53.6/1,000 population). (Persons of other races are excluded from this consideration because they constitute a smaller and much more heterogeneous group that is not directly comparable to the other populations.) Clearly, blacks as a group are at increased risk of premature death when compared with whites.

In accordance with the racial disparity in rates of premature mortality from all causes, blacks have higher YPLL rates than whites for all of the causes of mortality presented in Table V of the *MMWR*, with ratios ranging from 1.1 to 3.6 times the rates of the white population

FIGURE 3. Percent change in rates of years of potential life lost (YPLL)* before age 65, selected causes, United States, 1979-1984



*YPLL per 1,000 persons less than 65 years of age

(Table 5). The largest ratios, those causes for which blacks have the highest excess premature mortality, are sudden infant death syndrome, prematurity, and pneumonia and influenza.

Differences in the relative ranks of the leading causes of premature mortality can also be noted between races. For example, suicide/homicide was the leading cause of premature mortality for blacks in 1982 (13.6 YPLL/1,000 persons), but the fourth leading cause for whites (5.4/1,000 persons). The rates for unintentional injuries and malignant neoplasms, however, are more comparable (ranking first and second, respectively, for whites, and second and fourth for blacks).

Variations in YPLL by Sex

The rate of YPLL from all causes varies considerably for males and females. Males had a rate of 76.6 YPLL/1,000 population in 1982, nearly twice the rate for females (41.6/1,000 population). In addition, men had higher rates than women for all of the 12 leading causes of premature mortality, with YPLL rate ratios ranging from 1.1 to 3.5 (Table 6). The largest excess in male premature mortality is in intentional (suicide and homicide) and unintentional injuries, with ratios of 3.5 and 3.1, respectively. In contrast, the rates for malignant neoplasms and cerebrovascular diseases have male-to-female ratios of 1.1.

Similar to the differences in racial groups, the ranking of sex-specific YPLL rates by cause varies markedly by sex. As an illustration, although the rates for malignant neoplasms are similar for both sexes (9.2 YPLL/1,000 population for men and 8.5/1,000 for women), malignant neoplasms are the leading cause of premature mortality for women but only the fourth leading cause for men. This difference reflects the excess risk of premature mortality for men due to heart disease and unintentional and violent injuries.

Race- and Sex-Specific YPLL Rates

To define public health priorities further, race- and sex-specific YPLL rates can be determined. Black males had the highest rate of YPLL from all causes in 1982, followed by black women and white men; white women had the lowest rate (Figure 4). Black males had 3.5 times the risk of premature mortality of white women, while black women and white men had

TABLE 5. Rates of years of potential life lost (YPLL)* and YPLL rate ratios, by race, selected causes, United States, 1982

Cause of Mortality	Rates of YPLL		YPLL rate ratio (black/white)
	Blacks	Whites	
Suicide and homicide	13.6	5.4	2.5
Unintentional injuries	12.7	11.6	1.1
Heart disease	11.7	6.5	1.8
Malignant neoplasms	10.4	8.8	1.2
Prematurity	6.2	1.9	3.3
Congenital malformations	4.8	3.4	1.4
Sudden infant death syndrome	3.7	1.3	2.8
Cerebrovascular disease	2.9	0.8	3.6
Chronic liver disease	2.3	1.1	2.1
Pneumonia and influenza	2.0	0.6	3.3
Diabetes mellitus	1.0	0.5	2.0
Chronic obstructive pulmonary disease	0.9	0.5	1.8

*YPLL before age 65 per 1,000 persons less than 65 years a rate nearly twice that of white women. Black men have now been clearly identified as the group most at risk of premature death and, consequently, most in need of interventions to lower this risk.

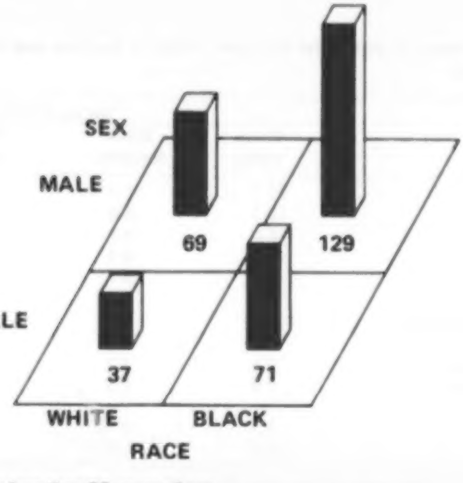
An even more striking difference can be noted in selected cause-specific YPLL rates (Table 7). Intentional injuries, particularly homicide, are identified as predominantly a condition affecting black males, with white males having the second highest rate. In contrast, the pattern for malignant neoplasms is more evenly distributed among all race/sex groups.

TABLE 6. Rates of years of potential life lost (YPLL)* and YPLL rate ratios, by sex, selected causes, United States, 1982

Causes of Mortality	Rates of YPLL		YPLL rate ratio (male/female)
	Males	Females	
Unintentional injuries	17.8	5.7	3.1
Suicide and homicide	10.1	2.9	3.5
Heart disease	9.7	4.4	2.2
Malignant neoplasms	9.2	8.5	1.1
Congenital malformations	3.8	3.3	1.2
Prematurity	2.8	2.0	1.4
Sudden infant death syndrome	2.0	1.3	1.5
Chronic liver disease	1.7	0.8	2.1
Cerebrovascular disease	1.3	1.2	1.1
Pneumonia and influenza	1.0	0.6	1.7
Chronic obstructive pulmonary disease	0.7	0.6	1.4
Diabetes mellitus	0.6	0.5	1.2

*YPLL before age 65 per 1,000 persons less than 65 years

FIGURE 4. Rates of years of potential life lost (YPLL)* before age 65, all causes, by race and sex, United States, 1982



*YPLL per 1,000 persons less than 65 years of age

PUBLIC HEALTH USES OF YPLL

YPLL is only one of a number of mortality measures that have broad public health applications. Yet this simple index of premature mortality can complement traditional methods of quantitating mortality, such as crude or age-adjusted death rates. The major strengths of YPLL are that it is simple to compute and comprehend and it effectively emphasizes deaths of younger persons, in contrast to usual mortality statistics, which are dominated by deaths of the elderly.

Geographic variations in premature mortality can also be a useful tool for health planning at the state or local level. A delineation of the pattern of YPLL rates among varied populations can provide an accurate description of the causes of premature mortality for those persons at highest risk of premature death. This type of analysis can be done on national data, such as presented in this document, or on local mortality data.

State-specific rates of YPLL can be used for planning and evaluating local public health interventions. Sex- and race-specific YPLL rates can, within the statistical limitations of population size, be used to target and monitor those populations at highest risk. Calculation of YPLL at the state level is presently being evaluated by CDC in cooperation with interested state health departments. For example, the importance of meningitis and unintentional injuries as causes of premature mortality in Alaska can be readily seen by the calculation of YPLL (CDC, unpublished data).

YPLL and YPLL rates can assist in the performance of three basic public health functions: the establishment of research and resource priorities, the surveillance of temporal trends in premature mortality, and the evaluation of the effectiveness of program interventions. Within certain limitations, YPLL can serve these functions at the national, state, or local level. Finally, quantifying YPLL for individual causes of mortality or for behavioral risk factors associated with a range of mortality consequences (e.g., tobacco or alcohol use) can provide a simple method to target health education efforts to sections of the general population most in need of public health interventions.

TABLE 7. Rates of years of potential life lost (YPLL*), by race and sex, selected causes, United States, 1982

	Rates of YPLL			
Causes of Mortality	Black males	Black females	White males	White females
Suicide and homicide	23.1	4.9	8.3	2.6
Unintentional injuries	19.4	6.5	7.7	5.5
Heart disease	15.0	8.5	9.1	3.8
Malignant neoplasms	11.6	9.4	9.0	8.5
Prematurity	7.3	5.1	2.2	1.6
Congenital malformations	5.1	4.5	3.6	3.1
Sudden infant death syndrome	4.4	3.1	1.7	1.0
Chronic liver disease	3.0	1.8	1.5	0.6
Cerebrovascular disease	3.1	2.7	1.1	1.0
Pneumonia and influenza	2.5	1.5	0.7	0.5
Chronic obstructive pulmonary disease	1.1	0.7	0.6	0.4
Diabetes mellitus	0.9	1.0	0.5	0.4

*YPLL before age 65 per 1,000 persons less than 65 years

References

1. Dempsey M. Decline in tuberculosis: death rate fails to tell entire story. Am Rev Tuberculosis 1947;56:157-64.
2. Greville TNE. Decline in tuberculosis: death rate fails to tell entire story [Comment on Dempsey's article]. Am Rev Tuberculosis 1948;57:417-9.
3. Dickinson FG, Welker EL. What is the leading cause of death? AMA Bulletin 1948;64:1-25.
4. Haenszel W. Standardized rate for mortality defined in units of lost years of life. Am J Public Health 1950;40:17-26.
5. Doughty JH. Mortality in terms of lost years of life. Can J Public Health 1951;42:134-41.
6. Logan WP, Benjamin B. Loss of expected years of life—a perspective view of changes between 1848-72 and 1952. Month Bull Min Health (Gr Britain) 1953;12:244-52.
7. Perloff JD, LeBailly SA, Kletke PR, Budetti PP, Connelly JP. Premature death in the United States: years of life lost and health priorities. J Public Health Policy 1984;5:167-84.
8. Stickle G. What priority, human life? Am J Public Health 1965;55:1692-8.
9. Romeder JM, McWhinnie JR. Potential years of life lost between ages 1 and 70: an indicator of premature mortality for health planning. Int J Epidemiol 1977;6:143-51.
10. Lalonde M. A new perspective on the health of Canadians: a working document. Ottawa, Canada: Department of National Health and Welfare, 1974.
11. CDC. Introduction to Table V: premature deaths, monthly mortality, and monthly physician contacts—United States. MMWR 1982;31:109-10, 117.
12. National Center for Health Statistics. Annual summary of births, marriages, divorces, and deaths, United States, 1984. Hyattville, Md.: Public Health Service, 1985. (Monthly Vital Statistics Report. Vol. 33, No. 13.) (DHHS publication no. (PHS) 85-1120).
13. U.S. Bureau of the Census. Current population reports, series P-25, No. 965. Estimates of the population of the United States, by age, sex, and race: 1980 to 1984. Washington, DC: U.S. Government Printing Office, 1985.
14. Jaffe HW, Hardy AM, Morgan WM, Darrow WW. The acquired immunodeficiency syndrome in gay men. Ann Intern Med, 1985;103:662-664.
15. CDC. Changes in premature mortality—United States, 1983-1984. MMWR 1986;35:27, 29-31.
16. CDC. Alcohol-associated premature mortality—United States, 1980. MMWR 1985;34:493-5.

NEWARK PUBLIC LIBRARY
NOV 17 1986
U. S. GOVT. PUBS.
DEPOSITORY 378

Director, Centers for Disease Control
James O. Mason, M.D., Dr.P.H.
Director, Epidemiology Program Office
Carl W. Tyler, Jr., M.D.
Supplement Editor
Linda Kay McGowan
Editor
Michael B. Gregg, M.D.
Assistant Editor
Karen L. Foster, M.A.

☆U.S. Government Printing Office: 1986-746-149/40024 Region IV

UNITED STATES GOVERNMENT PRINTING OFFICE
SUPERINTENDENT OF DOCUMENTS
Washington, D.C. 20402

OFFICIAL BUSINESS
Penalty for Private Use, $300

BULK RATE
POSTAGE & FEES PAID
GPO
Permit No. G-26

MMWR NEWAROOON DR 376 F 1
NEWARK PUBLIC LIBR
5 WASHINGTON ST
NEWARK NJ 07101

HHS Publication No. (CDC) 86-8017

Redistribution using indicia is illegal.

35/2SS

CENTERS FOR DISEASE CONTROL

MMWR

MORBIDITY AND MORTALITY WEEKLY REPORT

Vol. 35 / No. 2SS

CDC Surveillance Summaries

August 1986

NEWARK PUBLIC LIBRARY
NOV [illegible]
[illegible]
DEPOSITORY [illegible]

Contents

Patterns of Alcohol Consumption and Alcohol-Related Morbidity and Mortality

Abortion Surveillance: Preliminary Analysis— United States, 1982-1983

Trichinosis Surveillance, 1984

The National Occupational Health Survey of Mining

Trends in the Incidence of Endometrial and Ovarian Cancers

Ectopic Pregnancy in the United States, 1970-1983

U.S. DEPARTMENT OF HEALTH AND HUMAN SERVICES
PUBLIC HEALTH SERVICE
CENTERS FOR DISEASE CONTROL
ATLANTA, GEORGIA 30333

HE20.7009:

CDC Surveillance Summaries are published by the Epidemiology Program Office, Centers for Disease Control, Public Health Service, U.S. Department of Health and Human Services, Atlanta, Georgia 30333.

SUGGESTED CITATIONS

General:	Centers for Disease Control. *CDC Surveillance Summaries*, August 1986. *MMWR* 1986;35(No. 2SS).
Specific:	Centers for Disease Control. [Title of particular article/chapter.] In: *CDC Surveillance Summaries*, August 1986. *MMWR* 1986;35(No. 2SS):[inclusive page numbers].

Centers for Disease Control — James O. Mason, M.D., Dr.P.H., *Director*

This report was prepared by:

Epidemiology Program Office — Carl W. Tyler, Jr., M.D., *Director*

Michael B. Gregg, M.D., *Deputy Director for Communications*

Editorial Services — R. Elliott Churchill, M.A., *Chief*

Norma W. Strawn, *Writer-Editor*

Patsy H. Hurst, *Illustrator*

Roberta H. Rhodes, *Editorial Assistant*

Division of Surveillance and Epidemiologic Studies — Stephen B. Thacker, M.D., *Director*

Table of Contents

Foreword .... iiSS

History of CDC Surveillance Activities .... iiiSS

Data Sources .... ivSS

Surveillance Programs, CDC .... vSS

Patterns of Alcohol Consumption and Alcohol-Related Morbidity and Mortality
Ruth L. Berkelman, M.D., Mel Ralston, Joy Herndon, M.S., Marta Gwinn, M.D., Darryl Bertolucci, M.A., Mary Dufour, M.D., M.P.H. 1SS

Abortion Surveillance: Preliminary Analysis—United States, 1982-1983
Hani K. Atrash, M.D., M.P.H., Elaine P. Rhodenhiser, Carol J. R. Hogue, Ph.D., M.P.H., Jack C. Smith, M.S 7SS

Trichinosis Surveillance, 1984
Jeanette K. Stehr-Green, M.D., Peter M. Schantz, V.M.D., Ph.D., Emily M. Chisolm, M.P.H. 11SS

The National Occupational Health Survey of Mining
Dennis W. Groce, M.P.H., Wallace G. Carr, M.S., Frank J. Hearl, S.M. 17SS

Trends in the Incidence of Endometrial and Ovarian Cancers
Marta L. Gwinn, M.D., Nancy C. Lee, M.D., George L. Rubin, M.B.B.S. 23SS

Ectopic Pregnancy in the United States, 1970-1983
Hani K. Atrash, M.D., M.P.H., Joyce M. Hughes, Carol J.R. Hogue, Ph.D., M.P.H. 29SS

State and Territorial Epidemiologists and State Laboratory Directors .... Inside back cover

Foreword

The purpose of the *CDC Surveillance Summaries* is to make available the most current information on conditions of public health interest for which CDC has major responsibility. The *CDC Surveillance Summaries* provide detailed analysis of the most current available data obtained for CDC surveillance programs. These reports complement other data published by CDC in the *Morbidity and Mortality Weekly Report (MMWR)*, the *MMWR Annual Summary*, and various disease-surveillance reports. This volume contains epidemiologic information derived from surveillance forms, special investigations, and other sources of information collected at the state and national levels.

History of CDC Surveillance Activities

CDC has been actively involved in disease-surveillance activities since the formation of the Communicable Disease Center in 1946. The original scope of the National Surveillance Program included the study of malaria, murine typhus, smallpox, psittacosis, diphtheria, leprosy, and sylvatic plague. In 1954, a surveillance section was established within the Epidemiology Branch of CDC, primarily concerned with planning and conducting continuing surveillance and making periodic reports. National emergencies such as the Asian influenza pandemic and the discovery of Legionnaires' disease have prompted the involvement of CDC in new surveillance activities. Over the years the surveillance activities of CDC have expanded to include not only new areas in infectious disease but also programs in human reproduction, environmental health, chronic disease, risk reduction, and occupational safety and health. Ongoing evaluation of these programs has led to new methods of data collection and analysis and has prompted examination of how data are disseminated to the public health community.

In 1980 and 1981, a survey of CDC staff and state epidemiologists suggested that improved coordination of surveillance reports with the *MMWR* and the *MMWR Annual Summary* would facilitate timely publication; provide greater uniformity in the acquisition, evaluation, and reporting of surveillance data; and encourage use of these data. Several approaches to the development of a systematic process of disseminating disease-specific surveillance reports were considered. On the basis of considerations of timeliness, cost advantages, and editorial uniformity, a report published on a quarterly basis was recommended. Subsequent financial and personnel constraints have made it necessary to publish these reports less frequently.

The *CDC Surveillance Summaries* contain information more reflective of the detailed surveillance reports of the past. CDC hopes that the *Surveillance Summaries* will disseminate surveillance data on a regular schedule, improve the clarity of community public health information, and also produce a cost savings. Although the *CDC Surveillance Summaries* are published more often than once a year, they will typically contain annual data rather than interim data. The *MMWR Annual Summary* will complement rather than serve as the cumulative summary of the *Surveillance Summaries*.

Data Sources

Data on the reported occurrence of notifiable diseases are derived from reports supplied by the state and territorial departments of health and CDC program activities. These data are routinely published in the *MMWR* and compiled in final form in the *MMWR Annual Summary.*

CDC also maintains national surveillance programs for selected diseases—with the cooperation of state and local health departments and other federal agencies—and publishes detailed epidemiologic analyses periodically. Data appearing in the *CDC Surveillance Summaries* or in a surveillance report may not agree exactly with reports published in the *MMWR* because of differences in timing of reports or because of refinements in case definition. It should be noted that data collected for the *MMWR* and the more detailed data published by individual CDC programs are collected independently.

These data should be interpreted with caution. Some diseases that cause severe clinical illness and are associated with serious consequences are probably reported quite accurately. However, diseases that are clinically mild and infrequently associated with serious consequences are less likely to be reported. Additionally, subclinical cases are seldom detected except in the course of epidemic investigations or special studies. The degree of completeness of reporting is also influenced by the diagnostic facilities available, the control measures in effect, and the interests and priorities of state and local officials responsible for disease control and surveillance. Finally, factors such as the introduction of new diagnostic tests and the discovery of new disease entities may cause changes in disease reporting independent of the true incidence of disease. Despite these limitations, the data in these reports have proven to be useful in the analysis of trends.

Surveillance Programs, CDC

Surveillance program	Responsible unit	Most recent report/summary*
Abortion	Division of Reproductive Health Center for Health Promotion and Education	1986 (SS 35/2) (data from 1982-1983))
Alcohol	Division of Surveillance and Epidemiologic Studies Epidemiology Program Office	1986 (SS 35/2) (data from 1980-1983)
Behavioral risk factors	Division of Nutrition Center for Health Promotion and Education	1984 (SS 33/1) (data from 1981-1983)
Berylliosis cohorts: registry of disease and exposure	Surveillance Branch Division of Surveillance, Hazard Evaluations, and Field Studies National Inst. for Occup. Safety & Hlth.	March 1983 (data from 1951-1980)
Biologics	Data Management Branch Division of Immunization Center for Prevention Services	December 1982 (1982 data)
Botulism	Enteric Diseases Branch Division of Bacterial Diseases Center for Infectious Diseases	May 1979 (data from 1899-1977)
Brucellosis	Bacterial Zoonoses Activity Division of Bacterial Diseases Center for Infectious Diseases	June 1979 (1978 data)
Cancers, endometrial and ovarian	Epidemiologic Studies Branch Division of Reproductive Health Center for Health Promotion and Education	1986 (SS 35/2) (data from 1973-1981)
Coal workers' pneumoconiosis	Epidemiological Investigations Branch Division of Respiratory Disease Studies National Inst. for Occup. Safety & Hlth.	1985 (SS 34/1) (data from 1970-1981)
Congenital malformations	Division of Birth Defects and Developmental Disabilities Center for Environmental Health	September 1985 (data from 1981-1983)
Dengue	Dengue Branch Division of Vector-Borne Viral Diseases Center for Infectious Diseases	1985 (SS 34/2) (data from 1983-1984)
Diabetes	Division of Diabetes Control Center for Prevention Services	June 1979 (1978 data)

---

*Publications denoted by "SS" appeared in issues of *CDC Surveillance Summaries*. Other reports listed can be obtained by contacting the responsible administrative unit listed.

Surveillance Programs, CDC

Surveillance program	Responsible unit	Most recent report/summary*
Diphtheria	Surveillance, Investigations, and Research Branch Division of Immunization Center for Prevention Services	July 1978 (data from 1971-1975)
Ectopic pregnancy	Division of Reproductive Health Center for Health Promotion and Education	1986 (SS 35/2) (data from 1970-1983)
Encephalitis	Arbovirus Reference Branch Division of Vector-Borne Viral Diseases Center for Infectious Diseases	May 1981 (1978 data)
Enterovirus	Respiratory and Enterovirus Branch Division of Viral Diseases Center for Infectious Diseases	November 1981 (data from 1970-1979)
Fifteen leading causes of death in the U.S., 1978	Health Analysis and Planning for Preventive Services Center for Prevention Services	September 1982 (1978 data)
Foodborne disease	Enteric Diseases Branch Division of Bacterial Diseases Center for Infectious Diseases	1986 (SS 35/1) (1982 data)
Gonorrhea	Division of Sexually Transmitted Diseases Center for Prevention Services	1984 (SS 33/4) (data from 1983-1984)
Hepatitis	Hepatitis Branch Division of Viral Diseases Center for Infectious Diseases	1985 (SS 34/1) (data from 1982-1983)
Homicide	Violence Epidemiology Branch Office of the Director Center for Health Promotion and Education	May 1983 (SS 32/2) (data from 1970-1978)
Hysterectomy	Epidemiologic Studies Branch Division of Reproductive Health Center for Health Promotion and Education	1986 (SS 35/1) (data from 1981-1982)
Influenza	Influenza Branch Division of Viral Diseases Center for Infectious Diseases	July 1984 (data from 1983-1984)
Lead poisoning in workers	Surveillance Branch Division of Surveillance, Hazard Evaluations, and Field Studies National Inst. for Occup. Safety & Hlth.	April 1983 (data from 1976-1980)

---

*Publications denoted by "SS" appeared in issues of *CDC Surveillance Summaries*. Other reports listed can be obtained by contacting the responsible administrative unit listed.

Surveillance Programs, CDC

Surveillance program	Responsible unit	Most recent report/summary*
Leprosy	Respiratory and Special Pathogens Branch Division of Bacterial Diseases Center for Infectious Diseases	April 1976 (data from 1971-1973)
Leptospirosis	Bacterial Zoonoses Activity Division of Bacterial Diseases Center for Infectious Diseases	August 1979 (1978 data)
Malaria	Malaria Branch Division of Parasitic Diseases Center for Infectious Diseases	September 1985 (1984 data)
Maternal mortality	Division of Reproductive Health Center for Health Promotion and Education	1984 (SS 33/1) (data from 1974-1978)
Measles	Surveillance, Investigations, and Research Branch Division of Immunization Center for Prevention Services	September 1982 (data from 1977-1981)
Mumps	Surveillance, Investigations, and Research Branch Division of Immunization Center for Prevention Services	July 1984 (data from 1977-1982)
National electronic injury surveillance system	Safety Surveillance Branch Division of Safety Research National Inst. for Occup. Safety & Hlth.	May 1983 (SS 32/2) (1982 data)
National Occupational Hazard Survey (NOHS)	Surveillance Branch Division of Surveillance, Hazard Evaluations, and Field Studies National Inst. for Occup. Safety & Hlth.	NIOSH Technical Report DHHS (NIOSH) Pub. No. 83-117
National Occupational Health Survey of Mining	Division of Respiratory Disease Studies National Inst. for Occup. Safety & Hlth.	1986 (SS 35/2) (data through 1985)
Nosocomial infections	National Nosocomial Infections Surveillance System Hospital Infections Program Center for Infectious Diseases	1986 (SS 35/1) (1984 data)
Nutrition	Division of Nutrition Center for Health Promotion and Education	November 1982 (1980 data)

---

*Publications denoted by "SS" appeared in issues of *CDC Surveillance Summaries*. Other reports listed can be obtained by contacting the responsible administrative unit listed.

Surveillance Programs, CDC

Surveillance program	Responsible unit	Most recent report/summary*
Occupational characteristics of disabled workers	Surveillance Branch Division of Surveillance, Hazard Evaluations, and Field Studies National Inst. for Occup. Safety & Hlth.	July 1980 (data from 1969-1978)
Occupational hazard surveillance	Surveillance Branch Division of Surveillance, Hazard Evaluations, and Field Studies National Inst. for Occup. Safety & Hlth.	1985 (SS 34/2) (data from 1970-1982)
Occupational injuries among loggers	Safety Surveillance Branch Division of Safety Research National Inst. for Occup. Safety & Hlth.	August 1983 (SS 32/3) (data from 1969-1974)
Occupational injuries in the meatpacking industry	Safety Surveillance Branch Division of Safety Research National Inst. for Occup. Safety & Hlth.	1985 (SS 34/1) (data from 1976-1981)
Occupational mortality in Washington State	Surveillance Branch Division of Surveillance, Hazard Evaluations, and Field Studies National Inst. for Occup. Safety & Hlth.	DHHS (NIOSH) Pub. No. 83-116 (data from 1950-1979)
Pediatric nutrition	Division of Nutrition Center for Health Promotion and Education	1983 (SS 32/4) (1982 data)
Pelvic inflammatory disease	Division of Sexually Transmitted Diseases Center for Prevention Services	1983 (SS 32/4) (data from 1965-1982)
Plague	Plague Branch Division of Vector-Borne Viral Diseases Center for Infectious Diseases	1985 (SS 34/2) (1984 data)
Poliomyelitis	Surveillance, Investigations, and Research Branch Division of Immunization Center for Prevention Services	December 1982 (data from 1980-1981)
Psittacosis	Bacterial Zoonoses Activity Division of Bacterial Diseases Center for Infectious Diseases	February 1983 (SS 32/1) (1979 data)
Rabies	Viral and Rickettsial Zoonoses Branch Division of Viral Diseases Center for Infectious Diseases	1985 (SS 34/1) (1983 data)

---

*Publications denoted by "SS" appeared in issues of *CDC Surveillance Summaries*. Other reports listed can be obtained by contacting the responsible administrative unit listed.

Surveillance Programs, CDC

Surveillance program	Responsible unit	Most recent report/summary*
Reye syndrome	Epidemiology Office Division of Viral Diseases Center for Infectious Diseases	1984 (SS 33/3) (1983 data)
Rickettsial disease (RMSF, murine typhus, Q fever, endemic typhus)	Viral and Rickettsial Zoonoses Branch Division of Viral Diseases Center for Infectious Diseases	May 1981 (1979 data)
Rocky mountain spotted fever	Viral and Rickettsial Zoonoses Branch Division of Viral Diseases Center for Infectious Diseases	1984 (SS 33/3) (data from 1981-1983)
Rubella	Surveillance, Investigations, and Research Branch Division of Immunization Center for Prevention Services	1984 (SS 33/4) (1983 data)
Salmonella	Enteric Diseases Branch Division of Bacterial Diseases Center for Infectious Diseases	December 1982 (1980 data)
Sentinel health event (occupational) (SHE)	Surveillance Branch Division of Surveillance, Hazard Evaluations, and Field Studies National Inst. for Occup. Safety & Hlth.	September 1983
Summer mortality	Division of Environmental Hazards and Health Effects Center for Environmental Health	February 1983 (SS 32/1) (data from 1979-1981)
Surgical sterilization	Epidemiologic Studies Branch Division of Reproductive Health Center for Health Promotion and Education	August 1983 (SS 32/3) (data from 1979-1980)
Toxic-shock syndrome	Respiratory and Special Pathogens Branch Division of Bacterial Diseases Center for Infectious Diseases	1984 (SS 33/3) (data from 1960-1984)
Trichinosis	Helminthic Diseases Branch Division of Parasitic Diseases Center for Infectious Diseases	1986 (SS 35/2) (1984 data)

---

*Publications denoted by "SS" appeared in issues of *CDC Surveillance Summaries*. Other reports listed can be obtained by contacting the responsible administrative unit listed.

Surveillance Programs, CDC

Surveillance program	Responsible unit	Most recent report/summary*
Tuberculosis	Division of Tuberculosis Control Center for Prevention Services	July 1985 (1984 data) TB Statistics: States & Cities
		November 1983 (1980 data) TB in the United States
U.S. immunization survey	Surveillance, Investigations, and Research Branch Division of Immunization Center for Prevention Services	April 1983 (data from 1979-1982)
Venereal disease	Division of Sexually Transmitted Diseases Center for Prevention Services	(1980 data) Sexually Transmitted Diseases Statistical Letter-No. 130
		(data from 1978-1979) STD Fact Sheet-Edition 35
Water-related disease outbreaks	Enteric Diseases Branch Division of Bacterial Diseases Center for Infectious Diseases	September 1984 (1983 data)

---

*Publications denoted by "SS" appeared in issues of *CDC Surveillance Summaries*. Other reports listed can be obtained by contacting the responsible administrative unit listed.

Patterns of Alcohol Consumption and Alcohol-Related Morbidity and Mortality

Ruth L. Berkelman, M.D.
Mel Ralston
Joy Herndon, M.S.
Marta Gwinn, M.D.
Division of Surveillance and Epidemiologic Studies
Epidemiology Program Office

Darryl Bertolucci, M.A.
Mary Dufour, M.D., M.P.H.
Division of Biometry and Epidemiology
National Institute on Alcohol Abuse and Alcoholism

Introduction

Alcohol misuse rivals tobacco use as the most preventable health problem in the United States, and the prevention of alcohol and drug abuse has been targeted as one of the 1990 Objectives for the Nation (*1*). In this article, we review trends in alcohol consumption and in mortality and hospitalization rates for selected alcohol-related diagnoses. These data indicate a slight decline since 1980 in alcohol consumption and in deaths and hospital admissions attributed explicitly to alcohol. More intensive surveillance efforts are needed to establish the true magnitude of alcohol-related health problems.

Methods

Consumption. Estimates on per-capita alcohol consumption are based on sales figures published by the Distilled Spirits Council of the United States, the Wine Institute, and the United States Brewers' Association, Inc., for the years 1974-1984 (*2-5*). Provisional data (based on the first 3 months of data for 1985) from the 1985 National Center for Health Statistics (NCHS) Health Interview Survey, administered to a national probability sample of U.S. residents 18 years of age or older, describe patterns of consumption during the year preceding the interview (*6*). Data from CDC's Behavioral Risk Factor Survey indicate the prevalence of 1) consumption of two or more drinks daily in the 14 days before the interview and 2) self-reported driving after "having had perhaps too much to drink" (unpublished data, CDC). This survey is conducted on a state-by-state basis, with 15 states reporting in 1984. Patterns of alcohol use among teenagers in the United States are reported from the annual High School Senior Survey conducted by the National Institute on Drug Abuse (*7*); each year's sample is drawn from all seniors in public and private high schools in the coterminous United States.

Health Events. Numbers of deaths explicitly attributed to alcohol-related causes were obtained from NCHS mortality tapes for the years 1980-1983. Years of potential life lost between birth and age 65 years were also calculated for the same causes of death (*8*). In addition, the NCHS multiple-cause mortality tapes for 1982 were examined for the presence of either alcohol dependence (International Classification of Diseases, 9th Revision [ICD-9] code 303) or alcohol abuse (ICD-9 code 305.0).

Numbers of hospitalizations for causes explicitly attributable to alcohol were obtained from the NCHS Hospital Discharge Survey; these data were based on a sample of discharges from short-stay nonfederal hospitals in the United States for the years 1980-1982.

Results

Consumption. From 1974 through 1984, there was a 0.7% decrease in per-capita consumption (among persons 14 years of age and older) of commercially produced beverage alcohol in the United States. This decrease was due entirely to a 15.4% decline in consumption of distilled spirits. Per-capita consumption of beer and wine rose 8.0% and 19.4%, respectively, over the same period. In 1984, 52.3% of beverage alcohol consumed in the United States was from beer, 34.3% from distilled spirits, and 13.4% from wine.

Provisional data from the 1985 NCHS Health Interview Survey (HIS) indicate that approximately 44% of Americans 18 years of age or older (33% of males and 43% of females) reported abstention from alcohol during the year immediately preceding interview. Fifty-two percent of respondents (63% of males and 42% of females) who reported drinking within the past year were defined by HIS as current drinkers (at least one drink within the preceding 2 weeks). Eleven percent of current drinkers (15% of males and 7% of females) reported daily alcohol use, and 8% (13% of males and 4% of females) reported having consumed five drinks or more on at least one occasion within the 2 weeks before the interview.

State-specific results of CDC's 1984 Behavioral Risk Factor Survey of adults 18 years of age and older indicate that the proportion of respondents who reported an average consumption of two or more drinks per day during the previous month ranged from 3% in Utah to 12% in Arizona (median, 8%). The proportion of respondents who reported driving after "having had perhaps too much to drink" ranged from 2% in South Carolina to 11% in Wisconsin (median, 5%).

According to the annual 1984 High School Senior Survey, 14% of respondents abstained from alcohol during the year preceding the interview, whereas 67% (71% of males and 63% of females) reported current use (within the preceding 30 days). Five percent of current drinkers (7% of males and 3% of females) reported daily alcohol use. The number of current users among high school seniors declined from 78% to 67% between 1978 and 1984, and the prevalence of daily use fell from 7% to 5% during the same period.

The prevalence of binge drinking (five or more drinks on at least one occasion within the preceding 2 weeks) has remained relatively high among high school students. From 1975 to 1979, the number of respondents reporting binge drinking increased from 37% to 41% and remained at that level through 1983. In 1984, 39% of respondents (48% of males and 30% of females) reported binge drinking in the 2 weeks before the interview.

Health Events. An overall decline in deaths attributable entirely to alcohol-related causes was seen for all primary diagnoses for the years 1980-1983, with the exception of alcoholic cardiomyopathy; however, the trends were not consistent (Table 1). The decrease in alcohol-related mortality was also reflected in an overall decline of 14% in years of potential life lost for these causes of death (Table 2). Although alcohol dependence and alcohol abuse were listed as the primary cause of death in only 0.2% of all deaths in 1982, examination of NCHS multiple-cause mortality tapes showed that one or both of these diagnoses were present in 22,000 (1.1%) of the deaths that year. At short-stay nonfederal hospitals, there was no consistent trend in the number of admissions for selected alcohol-related diagnoses (Table 3), although there were 4% fewer such admissions in 1982 than in 1980. Admissions associated with alcohol abuse rose 28% between 1980 and 1982, but admissions associated with alcohol dependence and alcohol psychosis decreased by 5% and 13%, respectively.

Discussion

These data demonstrate a slight overall decline in alcohol consumption between 1974 and 1984. Although most authorities believe that consumption trends correlate with overall trends in cirrhosis mortality, such trends should not be used alone in estimating the magnitude of alcohol problems (*9*).

Several of the 1990 Objectives relate to reducing risk factors, including alcohol misuse, among adolescents 12-17 years old (*1*). Currently, information on this age group is available at the national level only for high school seniors; these data indicate that the number of abstainers in this group is increasing, with a concomitant decrease in the number of teenagers reporting daily use. Reports of binge drinking, however, have not decreased over the past 4 years, and the number of such reports among adolescents far surpasses that among adults 18 years of age and older.

In concordance with data on the prevalence of consumption and other risk factors, information on selected adverse alcohol-related events indicates a slow decline in deaths from

TABLE 1. Deaths attributed to selected alcohol-related causes,* United States, 1980-1983

ICD-9 code	Cause	Deaths			
		1980	1981	1982	1983
291	Alcohol psychosis	454	453	389	346
303	Alcohol dependence	4,350	4,207	3,914	4,002
305.0	Alcohol abuse	889	771	768	835
425.5	Alcoholic cardiomyopathy	650	647	664	711
571.0	Alcoholic fatty liver	1,166	951	961	1,027
571.1	Acute alcoholic hepatitis	794	785	687	725
571.2	Alcoholic cirrhosis of liver	9,166	8,567	7,893	7,571
571.3	Alcoholic liver damage	1,812	1,782	1,752	1,753
E860	Toxic effects of ethyl alcohol	385	384	412	341
TOTAL		19,666	18,547	17,440	17,311

*Includes only those causes that were 100% attributable to alcohol misuse

TABLE 2. Years of potential life lost (YPLL) attributed to selected alcohol-related causes,* United States, 1980-1983

ICD-9 code	Cause	YPLL			
		1980	1981	1982	1983
291	Alcohol psychosis	5,630	4,982	4,045	3,390
303	Alcohol dependence	51,952	49,310	44,889	46,149
305.0	Alcohol abuse	16,873	15,565	15,758	17,366
425.5	Alcoholic cardiomyopathy	7,660	6,862	7,000	7,455
571.0	Alcoholic fatty liver	21,155	16,195	16,870	18,180
571.1	Acute alcoholic hepatitis	11,735	11,005	9,995	9,935
571.2	Alcoholic cirrhosis of liver	92,977	86,202	78,125	73,462
571.3	Alcoholic liver damage	21,570	21,470	20,825	20,567
E860	Toxic effects of ethyl alcohol	7,351	6,033	8,152	6,230
TOTAL		236,903	217,624	205,659	202,734

*Includes only those causes of years of potential life lost that were 100% attributable to alcohol misuse alcohol-related causes since 1980. Trends in numbers of hospital admissions due to alcohol-related diagnoses are less consistent.

Although trends for deaths and hospital admissions may be described for diagnoses that are 100% attributable to alcohol, it is difficult to follow trends for the majority of alcohol-related adverse events (e.g., medical diagnoses such as pancreatitis and head and neck cancer, trauma such as child abuse, and burns). For most diagnoses, estimates of the proportion of cases related to alcohol misuse either are not available or they range widely (*10*); furthermore, how these proportions have changed over time is unknown. Many other hospital admissions and deaths undoubtedly are related to—but not reported as 100% attributable to—chronic or acute effects of alcohol use.

The problem of alcohol misuse is complex, involving not only the drinker, but also his or her family, friends, and community. Currently, data on alcohol misuse in the United States are limited in both quality and quantity. Most surveillance activities have focused on alcohol consumption and production and not on health outcomes, with the exception of cirrhosis and—more recently—traffic fatalities (*11,12*). Although such data have been useful in following trends, they have been of limited value in measuring the impact of alcohol misuse on the community and in comparing the magnitude of alcohol-related health problems with other health problems (e.g., smoking-related illness) for which health authorities are competing for limited resources for prevention.

Efforts to improve the ascertainment of alcohol-related mortality have included increased blood-alcohol testing of motor-vehicle fatalities (*12*) and documentation by autopsy of chronic alcoholism unreported on death certificates (*13*). A recent study of postservice mortality among Vietnam-era veterans documents the shortcomings of death certificates as tools for the surveillance of alcohol-related fatalities, both for natural and for injury-related deaths; for example, a clinical review panel recorded alcohol-related impairment of the victims of unintended fatal injuries seven times more often than the certifiers who completed the death certificates (D. Pollock, personal communication, CDC). Findings from these and other studies (*14*) indicate that the morbidity and mortality related to alcohol misuse have probably been grossly underestimated. Basic research is needed to show the association between alcohol

TABLE 3. Alcohol-related admissions* to short-stay nonfederal hospitals, United States, 1980-1982

ICD-9 code	Diagnosis	Admissions 1980	1981	1982
291	Alcohol psychosis	66,088	72,705	57,505
303	Alcohol dependence	438,909	445,532	416,538
305.0	Alcohol abuse	38,461	40,404	49,395
425.5	Alcoholic cardiomyopathy	1,435	3,560	2,184
571.0	Alcoholic fatty liver	578	310	721
571.1	Acute alcoholic hepatitis	12,460	10,157	11,195
571.2	Alcoholic cirrhosis of liver	19,616	25,775	19,168
571.3	Alcoholic liver damage	9,556	9,668	7,067
980.0	Toxic effects of ethyl alcohol	905	402	239
TOTAL		588,008	608,513	564,012

*Includes only those causes of admissions that were 100% attributable to alcohol misuse consumption and specific adverse health outcomes if effective prevention programs are to be developed and supported.

References

1. US Department of Health and Human Services. Promoting health/preventing disease: objectives for the nation. Washington, DC: Government Printing Office, 1980.
2. Kling W. Distilled Spirits Council of the United States: annual statistical review 1984/85. Washington, DC: Distilled Spirits Council of the United States, 1985.
3. Wine Institute. Wine Institute economic research report: 1984 wine industry statistical report. San Francisco: Wine Institute, 1985.
4. Katz PC, King PA, Emamali B, Dall'Acqua J. The brewing industry in the United States. Washington, DC: United States Brewers' Association, Inc, 1984.
5. National Institute on Alcohol Abuse and Alcoholism. U.S. apparent consumption of alcoholic beverages based on state sales taxation or receipt data, 1985 (unpublished data).
6. National Center for Health Statistics. ADVANCEDATA: provisional data from the health promotion and disease prevention supplement to the National Health Interview Survey: United States, January-March 1985. Hyattsville, Maryland: US Department of Health and Human Services, 1985; DHHS publication no. (PHS) 86-1250.
7. Johnston LD, O'Malley PM, Bachman JG. National Institute on Drug Abuse. Use of licit and illicit drugs by America's high school students 1975-1984. Michigan: University of Michigan Institute for Social Research, 1985.
8. CDC. Changes in premature mortality—United States, 1983-1984. MMWR 1986;35:27,29-31.
9. Wilson RA. Changing validity of the cirrhosis mortality—alcoholic beverage sales construct: U.S. trends, 1970-1977. J Stud Alcohol 1984;45:53-8.
10. Harwood HJ, Napolitano DM, Kristiansen PL, Collins JJ. Economic costs to society of alcohol and drug abuse and mental illness: 1980. Research Triangle Institute, North Carolina: ADAMHA, Office of Program Planning and Coordination, 1984.
11. CDC. Reported cirrhosis mortality—United States, 1970-1980. MMWR 1984;33:657-9.
12. National Center for Statistics and Analysis. Fatal accident reporting system 1984. Washington, DC: US Department of Transportation, National Highway Traffic Safety Administration, 1984.
13. Kircher T, Nelson J, Burdo H. The autnpsy as a measure of accuracy of the death certificate. N Engl J Med 1985;313:1263-9.
14. Nashold RD, Naor EM. Alcohol-related deaths in Wisconsin: the impact of alcohol on mortality. Am J Public Health 1981;71:1237-41.

Abortion Surveillance: Preliminary Analysis—United States, 1982-1983

Hani K. Atrash, M.D., M.P.H.
Elaine P. Rhodenhiser
Carol J. R. Hogue, Ph.D., M.P.H.
Jack C. Smith, M.S.
Division of Reproductive Health
Center for Health Promotion and Education

Introduction

In 1969, the Division of Reproductive Health, Center for Health Promotion and Education, CDC, initiated ongoing epidemiologic surveillance of abortion in the United States. The objectives of this surveillance are twofold: 1) to document the numbers and characteristics of women obtaining abortion and 2) to assist in efforts to eliminate preventable morbidity and mortality associated with abortion. This report is a preliminary analysis of legal abortions reported to CDC for the years 1982 and 1983. The number of states reporting specific abortion characteristics varies each year; therefore, temporal trends based on these summary data should be interpreted with caution.

Numbers and Characteristics of Women Obtaining Abortions

In 1983—for the first time since 1969, when CDC began abortion surveillance—the total number of legal abortions, the rate per 1,000 females 15-44 years of age, and the ratio per 1,000 live births all declined in comparison with reported values for the previous year.

The number of legal abortions reported to CDC from the 50 states and the District of Columbia increased from 1,300,760 in 1981 to 1,303,980 in 1982 and decreased to 1,268,987 in 1983 (Table 1). This 2.7% decrease in 1983 was preceded by a continuous decline in the percent increase in the reported number of legal abortions since 1976 (*1*). The increase between 1976 and 1977 was 9.2%, compared with a 0.25% increase from 1981 to 1982.

The national abortion ratio continued to decrease from a high of 359 legal abortions per 1,000 live births in 1980 to 358 in 1981, 354 in 1982, and 349 in 1983. The national abortion rate of 24 legal abortions per 1,000 women 15-44 years of age for 1981 and 1982 was less than it had been the previous year, and it dropped to 23 in 1983.

In 1982 and 1983, as in previous years, women obtaining abortions tended to be young, white, and unmarried, and to have had no live births. In 1982, 62.2% of women obtaining legal abortions were under 25 years of age, 68.5% were white, 78.0% were unmarried, and 57.8% had had no live births. In 1983, 61.8% were under 25 years of age, 67.6% were white, 78.6% were unmarried, and 57.1% had had no live births.

Examination of the national abortion surveillance system for 1982 and 1983 suggests that the reported lower abortion ratios and abortion rates reflect real phenomena rather than being artifacts of changes in the completeness of reporting. For 1982, in 25 reporting areas, the number of abortions exceeded the number reported for 1981; for 27 areas, fewer abortions were reported than for 1981. For 1983, in 14 areas the number of abortions exceeded the number reported for 1982; in 36 areas, fewer abortions were reported than for 1982; in two areas there was no change in the reported numbers. The sources of abortion data were identical in 1982 and 1983.

TABLE 1. Characteristics of women obtaining abortions, United States, selected years

Characteristics	1972	1976	1980	1981	1982	1983
Reported Number of Legal Abortions	586,760	988,267	1,297,606	1,300,760	1,303,980	1,268,987
Ratio*	180.1	312.0	359.2	358.4	354.3	348.7
Rate†	13	21	25	24	24	23
			Percentage Distribution§			
Age (years)						
≤ 19	32.6	32.1	29.2	28.0	27.1	27.1
20-24	32.5	33.3	35.5	35.3	35.1	34.7
≥ 25	34.9	34.6	35.3	36.7	37.8	38.2
Race						
White	77.0	66.6	69.9	69.9	68.5	67.6
Black and other	23.0	33.4	30.1	30.1	31.5	32.4
Marital Status						
Married	29.7	24.6	23.1	22.1	22.0	21.4
Unmarried	70.3	75.4	76.9	77.9	78.0	78.6
Number of Live Births‖						
0	49.4	47.7	58.4	58.3	57.8	57.1
1	18.2	20.7	19.5	19.7	20.3	20.7
2	13.3	15.4	13.7	13.7	13.9	14.2
3	8.7	8.3	5.3	5.3	5.1	5.2
≥ 4	10.4	7.9	3.2	3.0	2.9	2.8
Type of Procedure						
Curettage	88.6	92.8	95.5	96.1	96.4	96.8
Suction curettage	*65.2*	*82.6*	*89.8*	*90.4*	*90.6*	*91.1*
Sharp curettage	*23.4*	*10.2*	*5.7*	*5.7*	*5.8*	*5.7*
Intrauterine instillation	10.4	6.0	3.1	2.8	2.5	2.1
Hysterotomy/ hysterectomy	0.6	0.2	0.1	0.1	0.0¶	0.0¶
Other	0.5	0.9	1.3	1.0	1.0	1.1
Weeks of Gestation						
≤ 8	34.0	47.0	51.7	51.2	50.6	49.7
9-10	30.7	28.0	26.2	26.8	26.7	26.8
11-12	17.5	14.4	12.2	12.1	12.4	12.8
13-15	8.4	4.5	5.2	5.2	5.3	5.8
16-20	8.2	5.1	3.9	3.7	3.9	3.9
≥ 21	1.3	0.9	0.9	1.0	1.1	1.0

*Abortions per 1,000 live births

†Abortions per 1,000 females 15-44 years of age

§Excludes unknowns. Since the number of states reporting each characteristic varies from year to year, temporal comparisons should be made with caution

‖For years 1972 and 1976, data indicate number of living children

¶<0.05%

Methods of Abortion

As in previous years, curettage continued to be the primary method of abortion. It accounted for a higher proportion in 1982 and 1983 than in previous years, whereas lower proportions of abortions were performed by instillation, hysterectomy, and hysterotomy.

In both 1982 and 1983, approximately 96% of all legal abortions were done by curettage, whereas approximately 2% were done by instillation, and less than 0.05% were done by hysterectomy and hysterotomy. About half of the legal abortions were performed in the first 8 weeks of gestation, and approximately 90% were done during the first 12 weeks of gestation.

Abortion-Related Mortality

CDC investigates all reports of abortion-related deaths. Late reports of confirmed abortion-related deaths are used to update data from previous years. Past experience shows that 64% of abortion-related deaths are identified and investigated within 12 months of the death and 96% within 3 years.

The number of legal-abortion-related deaths increased from seven in 1981 to 11 in 1982. The legal-abortion-related mortality rate in 1982 was also higher than that in 1981. The death-to-case rate for legal abortion in 1982 was 0.8 per 100,000, compared with 0.5 in 1981. The death-to-case rate has been generally decreasing since 1972, the year CDC began abortion mortality surveillance. However, increases in the abortion-related mortality rate have occurred twice previously—in 1977 (17 deaths, or 1.6 deaths per 100,000 abortions, in 1977, compared with 11 deaths, or 1.1 deaths per 100,000 abortions, in 1976) and in 1979 (18 deaths, or 1.4 deaths per 100,000 abortions, in 1979, compared with nine deaths, or 0.8 deaths per 100,000 abortions, in 1978).

Mortality data for 1983 are not presented in this report, since they may not be complete.

Reference

1. CDC. Abortion surveillance 1981. Atlanta: CDC, 1985.

Trichinosis Surveillance, 1984

Jeanette K. Stehr-Green, M.D.
Peter M. Schantz, V.M.D., Ph.D.
Emily M. Chisolm, M.P.H.
Helminthic Diseases Branch
Division of Parasitic Diseases
Center for Infectious Diseases

Introduction

Trichinosis, infection with *Trichinella spiralis*, continues to be a public health problem in the United States. Although fewer than 100 cases a year are now being reported, many mild or asymptomatic infections are undetected or misdiagnosed unless they are related epidemiologically to more severe cases (*1*). We believe that the reported cases represent only a fraction of the total number of cases that occur each year. With a stable surveillance system, however, cases that are reported can be useful in monitoring trends in the incidence of the disease, in initiating outbreak investigations, and in identifying groups at high risk for the infection.

Materials and Methods

State health departments report new cases of trichinosis by week to the National Morbidity Reporting Service. Supplemental epidemiologic information is submitted by the reporting state on Surveillance Case Report forms (CDC 54.7—Rev.7-81) to the Division of Parasitic Diseases (DPD), Center for Infectious Diseases (CID), CDC. Additional cases are identified through reported results of trichinosis serologic tests performed by the Helminthic Diseases Branch, DPD, CID, CDC, and through investigations conducted by the DPD staff.

Criteria for inclusion as a case:

1. *Trichinella*-positive muscle biopsy with clinical symptoms compatible with trichinosis (including eosinophilia, fever, myalgia, and periorbital edema),
2. Positive serologic test for trichinosis, or
3. Compatible symptoms (as above) in a patient with a history of ingesting meat known to contain *Trichinella* larvae.

Cases reported by the states but not characterized by written surveillance reports or not fitting the case definition are not included in this report.

Results

In 1984, 65 cases of trichinosis were reported to CDC. Thirteen states reported at least one case; however, 75% of the cases occurred in five states (California, Connecticut, Massachusetts, New Jersey, and Texas), and over 60% of the cases occurred in the Middle Atlantic and New England states (Table 1). For the third year in a row, New Jersey reported the largest number of cases. The states with the highest 5-year mean trichinosis incidence for the period 1980-1984 were Alaska (11.0 cases/million population), Rhode Island (7.8), Connecticut (3.9), New Jersey (2.7), and Vermont (2.0). Moderately high mean incidence was reported in Hawaii (1.5), Louisiana (1.3), and Massachusetts (1.2). No cases were reported in the 5-year period in Alabama, Arizona, Arkansas, Florida, Georgia, Iowa, Kentucky, Michigan, Minnesota, Mississippi, Montana, Nebraska, New Mexico, North Dakota, Oklahoma, South Carolina, South Dakota, Tennessee, Wisconsin, and Wyoming. The remaining states reported between 0.1 and 1.0 case/million population.

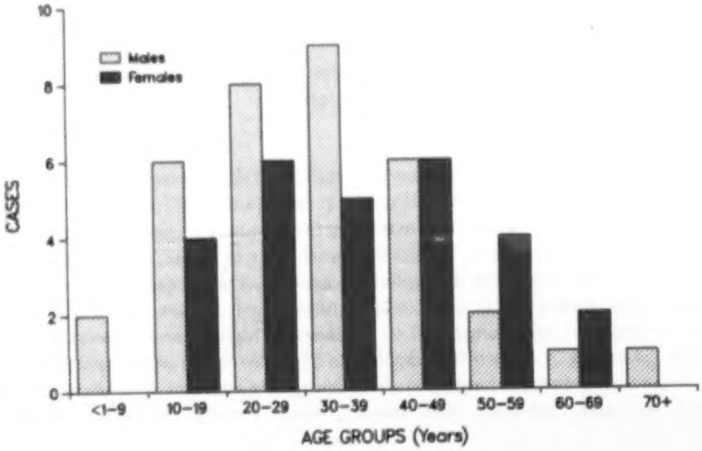
Of the 65 cases reported in 1984, 56.9% occurred in males and 43.1% in females. The mean age of patients was 34.2 years, with a range of 3-73 years. The age distribution by sex was similar (Figure 1).

The only consistent seasonal pattern for trichinosis in the United States has been a peak in December and January, related to consumption of homemade pork sausage during the Christmas holidays. Although a similar holiday peak was not evident in 1984, 12 cases reported in May were associated with kielbasa (Polish sausage) prepared by a Hungarian club for Easter (Figure 2).

TABLE 1. Trichinosis cases, by state, United States, 1984

State	Cases	(%)	Rate per million population
Alaska	3	(4.6)	7.5
California	8	(12.3)	0.3
Connecticut	7	(10.8)	2.3
Hawaii	1	(1.5)	1.0
Maine	1	(1.5)	0.9
Massachusetts	6	(9.3)	1.0
New Jersey	17	(26.2)	2.3
New York	4	(6.2)	0.2
North Carolina	1	(1.5)	0.2
Pennsylvania	4	(6.2)	0.3
Texas	11	(16.9)	0.7
Utah	1	(1.5)	0.7
Virginia	1	(1.5)	0.2
Total	**65**	(100.0)	

FIGURE 1. Trichinosis cases, by age group and sex, United States, 1984



All but four of the patients reported at least one of the common signs and symptoms of trichinosis: 92.6% had fever (50 of the 54 persons for whom this symptom was known), 74.5% (41/55) had periorbital edema, 96.3% (52/54) had myalgia, and 95.9% (47/49) had eosinophilia. Most of the patients recovered; however, one patient died, and the outcomes for six were unknown.

The mean incubation period for the 29 cases for which the dates of consumption of incriminated meat and onset of symptoms were available was 8.1 days and ranged from 1 to 34 days. Diagnosis of trichinosis was confirmed by serologic tests in 34 cases (97.1% of those in which serologic testing was done). Muscle biopsy was performed on 22 patients, and in 19 (86.4%) instances the results were positive.

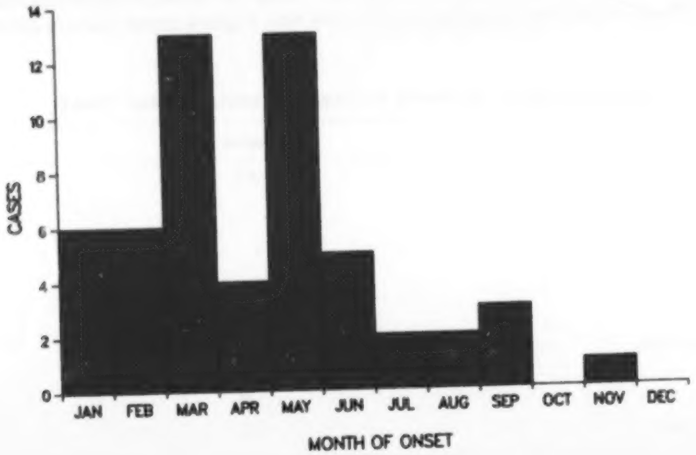
The infective meat item was identified in 60 of 65 cases; however, in only 18 cases (of which 12 were from the same outbreak) was the suspected food item examined for *Trichinella* larvae. All items were positive. Pork was incriminated in 53 (88.3%) of the cases and bear meat in six (10%). Sausage was the form of pork most frequently implicated (48.9%) (Table 2).

The method of meat preparation was identified in 50 cases; in 18 (36%) of these the meat was eaten raw, in 10 (20%) it was boiled, in seven (14%) it was roasted on an open fire, and in six (12%) it was fried. In the remaining nine cases, various methods of preparation were used. In 22 cases (40% of those in which a source was reported) the implicated meat was obtained from a supermarket, butcher shop, or other commercial outlet. In 11 (20%) cases it was obtained directly from the farm, and in seven (12.7%) the meat (feral swine and bear) was obtained through hunting.

Common-Source Outbreaks

Eight common-source outbreaks involving 40 people were reported to CDC in 1984. Two of the larger outbreaks were investigated thoroughly. The first involved a community of Laotians living in Amarillo, Texas. During March and April, 12 individuals from five unrelated

FIGURE 2. Trichinosis cases, by month of onset, United States, 1984



households developed symptoms of trichinosis after eating pork from pigs purchased separately from a small local pig farm. In six cases the diagnosis was confirmed by serologic testing. Five persons were hospitalized; they all recovered. Pork from the pigs was unavailable for examination, and investigation of the farm was unrevealing (*2*).

The second outbreak occurred in New Jersey after the sale of kielbasa made by a local Hungarian club for Easter. Of the 44 individuals exposed to the pork, 12 developed symptoms suggestive of trichinosis. The consumption of raw pork was associated with a relative risk of 5.7. Two patients were hospitalized, and one developed cardiac and neurologic complications. Examination of samples of the sausage revealed *Trichinella* larvae. In addition to the persons who ate kielbasa made by the club, five persons who purchased kielbasa from another source developed symptoms of trichinosis. Investigation revealed that the pork used to make the second kielbasa was obtained from the same meat-packing company as the pork used in the kielbasa sold by the club.

Discussion

In 1983, only 30 cases of trichinosis were reported in the United States, by far the fewest ever recorded (*3*). In 1984, more than twice as many cases were reported; there was one death. No apparent change in the surveillance system could account for this increase in cases. The increase may have been due in part to the large number of common-source outbreaks. In previous years, the majority of cases were associated with common sources, with years of peak incidence, such as 1969 and 1975, coinciding with an unusually large number of such cases (*4*). In 1983, only six cases were associated with common sources, compared with the 40 reported in 1984. No doubt, outbreak investigations allow for the detection and diagnosis of cases that would go undiagnosed and therefore unreported if they occurred as individual cases.

As in previous years, a large number of the reported cases occurred in the Middle Atlantic and New England states, a finding that has been associated with high concentrations of ethnic groups whose culinary preferences include raw pork (*5*). However, recent studies of selected slaughterhouses have revealed the widespread occurrence of infection in swine in these regions: 0.58% of the pigs examined from slaughterhouses in the Middle Atlantic states and 0.73% of those from slaughterhouses in the New England states were found to be infect-

TABLE 2. Trichinosis cases, by source of infection, United States, 1984

Food		Cases	(%)
Pork		53	(88.3)
Wild boar	1		
Sausage	23		
Chops	3		
Roast	6		
Ham	1		
Bacon	1		
Other	12		
Unspecified	6		
Bear		6	(10.0)
Ground beef		1	(1.7)

ed with *Trichinella* (*6,7*), surpassing the national estimate of 0.1% (*8*). These findings were attributed to the swine management practices of the small and/or part-time hog producers commonly found in these regions. The relative importance of these findings, in the light of culinary preferences of ethnic groups in these areas, needs further investigation.

Although the number of reported cases of trichinosis in the United States has decreased dramatically, trichinosis surveillance continues to be of interest. Specific subpopulations are at an increased risk for infection with trichinosis, traditionally Eastern European groups such as those of Italian, German, and Polish descent. As a result of trichinosis surveillance activities, Southeast Asian immigrants have been found to be a high-risk group, and public health measures have been directed accordingly. In addition, new cases of trichinosis in humans have been an impetus for action by the National Pork Producers Council to develop a national strategy for the control of trichinosis in swine.

References

1. Pawlowski ZS. Clinical aspects in man. In: Campbell WC, ed. *Trichinella* and trichinosis. New York: Plenum Press, 1983:367-401.
2. CDC. Trichinosis—Texas. MMWR 1984;33:517-8.
3. CDC. Trichinosis surveillance, 1983. In: CDC surveillance summaries. MMWR 1984;33(4SS): 17SS-23SS.
4. Schantz PM, Juranek DD, Schultz MG. Trichinosis in the United States, 1975: increase in cases attributed to numerous common-source outbreaks. J Infect Dis 1977;136:712-5.
5. Schantz PM. Trichinosis in the United States — 1947-1981. Food Tech March 1983:83-6.
6. Schad GA, Leiby DA, Duffy CH, Murrell KD. Swine trichinosis in New England slaughterhouses. Am J Vet Res 1985;46:2008-10.
7. Schad GA, Kelly M, Leiby DA, Blumrick K, Duffy CH, Murrell KD. Swine trichinosis in mid-Atlantic slaughterhouses: possible relationship to hog marketing systems. Prev Vet Med 1985;3:391-9.
8. Zimmermann WJ, Zinter DE. The prevalence of trichiniasis in swine in the United States, 1966-70. HSMHA Health Rep 1971;86:937-45.

The National Occupational Health Survey of Mining

Dennis W. Groce, M.P.H.
Wallace G. Carr, M.S.
Frank J. Hearl, S.M.
Division of Respiratory Disease Studies
National Institute for Occupational Safety and Health
Morgantown, West Virginia

Introduction

Surveillance for occupational safety and health problems has historically lagged behind that for infectious diseases. Adequate surveillance methods are essential in assessing the scope of health problems and in assigning priorities for intervention strategies; therefore, in recent years the National Institute for Occupational Safety and Health (NIOSH) has placed heavy emphasis on upgrading occupational health surveillance. Since direct surveillance of exposed workers has been shown to be a useful way to assess the prevalence of disease and to monitor trends, in 1984 NIOSH began an extensive survey of potential occupational exposures found at mine sites in the United States.

The National Occupational Health Survey of Mining (NOHSM) is similar to the National Occupational Hazard Survey that NIOSH conducted during 1972-1974 and the follow-up National Occupational Exposure Survey conducted during 1981-1982, both of which, however, involved only general-industry worksites. NOHSM not only will identify potential exposures related to occupational lung diseases, which head the NIOSH list of 10 leading work-related diseases and injuries (*1*), but also will identify potential exposures that may lead to other conditions on the list. The present plans for NOHSM call for five segments of mine surveys, to last approximately 1 year each, with an expected completion date of 1989. This report describes the purposes of NOHSM and the methods used. Results will be published in subsequent articles, as they become available.

NOHSM was developed in response to the U.S. Federal Mine Safety and Health Amendment Act of 1977, which requires the Secretary of Health, Education, and Welfare (now Health and Human Services) to "determine for each toxic material or harmful physical agent . . . used or found in a mine . . . whether such material or agent is potentially toxic at the concentrations in which it is used or found in a mine." As the lead federal agency for research in occupational safety and health, NIOSH was assigned responsibility for making this determination. To comply with the 1977 Act, NIOSH began in 1978 a review of information collected by several agencies (primarily the Mine Safety and Health Administration [MSHA]) on potentially toxic substances that exist on U.S. mine properties. It issued a report in 1979 showing a ". . . paucity of exposure data for certain agents, and the absence of comprehensive information on the many substances used or found in mines." As a result, NIOSH began planning for an in-depth survey of all substances found on mine properties. The study design was developed in the period 1980-1983, and NOHSM was approved for field work in March 1984.

Data from NOHSM will be used primarily to supply MSHA with information for 1) setting regulatory priorities and writing improved health standards, 2) improving compliance with existing standards, and 3) identifying research needs and priorities. NIOSH will also use the data to set priorities for its mining-related research and to provide supporting information for specific mining-related NIOSH projects. Other interested parties, including government agencies, universities, labor unions, and individual workers, are expected to request ingredient information for trade-name products found in the mining industry, along with supporting information for research efforts, compensation claims, and the instruction of students. With the exception of trade secrets, NIOSH will provide the data requested.

Survey Design and Sample Selection

The mining industry consists of specific mines and mills that produce commodities (e.g., asbestos, coal, potash) or carry out processes (e.g., crushing, breaking). NIOSH accepts the commodity or process designation that MSHA has established for each mine or mill. NOHSM will consist of a series of segments, each approximately 1 year in duration, with a different set of commodities to be studied in each segment. The five segments now planned will enable researchers to estimate 1) the number of miners potentially exposed to health hazards, 2) the number of mines with health hazards and with various occupational health facilities, and 3) the number of such health hazards and health facilities, arranged by Standard Industrial Classification and by other domains to be selected after the survey is completed.

Mines to be surveyed as a part of NOHSM are selected from a list of all mines that report quarterly to MSHA. Selection is based on average yearly employment, Standard Industrial Classification code, geographic area, and current activity in the mining of specified commodities. The 72 commodities to be studied in NOHSM are divided into 103 geographic strata based on differences in mineral composition and mining methods. Commodities surveyed in the first segment are listed in Table 1; those being surveyed in the second segment are shown in Table 2.

TABLE 1. Commodities* surveyed in the first segment of the National Occupational Health Survey of Mining

Aluminum/alumina
Aplite
Asbestos
Beryl
Boron minerals
Gemstones
Gilsonite
Gypsum
Leonardite
Lode, placer gold
Magnesite
Mercury
Metal ores†
Nonmetal minerals†
Perlite
Potash
Rare earths
Salt (evaporated)
Salt (rock)
Sandstone (crushed and broken)
Silver
Sodium compounds
Trona
Vermiculite

*Commodity terminology of the Mine Safety and Health Administration

†Not elsewhere classified

TABLE 2. Commodities* being surveyed in the second segment of the National Occupational Health Survey of Mining

Coal (bituminous)
Clay
Granite, crushed and broken
Granite, dimension
Manganese
Slate, crushed and broken
Slate, dimension
Stone, crushed and broken†
Stone, dimension†

*Commodity terminology of the Mine Safety and Health Administration

†Not elsewhere classified

Mines are selected according to two categories: 1) self-representing units for which the data collected will apply only to the individual mine and 2) probability samples for which the data may be projected to other similar mine sites. A site is designated as a self-representing unit if it 1) is one of the three largest mines or mills in the stratum, 2) has large employment relative to the rest of the stratum, or 3) is one of only a small number of mines in that stratum. Probability samples are selected to represent a given percentage of each stratum, with the probability of selection being proportional to the number of workers. Objective rules govern the selection process for probability samples to ensure that the samples are representative.

Field Protocol

Field activities for NOHSM are carried out by a team of six surveyors; normally each site visit is conducted by one surveyor. The initial six surveyors included three mining engineers, one mineral-processing engineer, and two industrial hygienists; the current team is composed of four mining engineers, one chemical engineer, and one industrial hygienist. Surveyors travel 100% of their duty time, and an office-support staff of three full-time and several part-time employees is based at the Appalachian Laboratory for Occupational Safety and Health (ALOSH) in Morgantown, West Virginia.

After each site is selected from the MSHA list, its management is contacted by telephone to verify that the mine is active and that the commodity designation is correct. A letter of notification of selection for NOHSM is then sent to the management of the mine and to the official miners' representative who is registered with MSHA. The survey is usually scheduled 1-2 months after notification. Surveyors are responsible for scheduling the surveys, and whenever possible they do this at a time mutually agreeable to NIOSH and to the mine management. Once a starting date has been established, NIOSH sends letters announcing plans for the survey to the management at the mine site, any corporate headquarters, the miners' representative, MSHA, the Bureau of Mines, appropriate state agencies, and the NIOSH Regional Offices.

Each site survey includes a questionnaire and walk-through observations. The questionnaire, which takes about 30 minutes to complete, is designed to record information on a company's occupational health practices and to include related descriptive information. The walk-through survey takes place in two phases: an inventory and a worksite survey. For the inventory, the surveyor examines centralized storage areas on the property and identifies every health-related trade-name product and generic chemical present. This includes potentially hazardous products such as mill reagents, fuels, lubricants, paints, cleaning agents, and welding rods. The surveyor must record the exact name of each product as labeled and the supplier's name and address, as well as the mine management's best estimate of the amount of the product used during the preceding 12 months. Products found on the site but not used during this period are recorded as having zero usage. For the worksite survey, the surveyor observes every worksite on the mine property and all activities of the workers to determine any health-related potential exposures. In addition to recording the toxic agents suggested above, the surveyor also records potential exposures to welding and combustion products, metals, dusts, excessive noise, musculoskeletal overloads, and a variety of ergonomic factors. For each potential exposure, the surveyor must determine its relative duration, the controls intended for that agent, and the associated number of employees by sex. This is strictly an observational exercise, and the term "potential exposure" is used to indicate that an agent is present but that NIOSH has not documented its level with any environmental monitoring technique.

After each survey, the surveyor codes the observations in a precise format and sends the coded data to ALOSH for computer processing and the generation of reports. At ALOSH, each set of survey data received from the field is keyed to magnetic tape, which is electronically edited to be sure that the data have been properly formatted. The computer editing also checks the chemicals mentioned in the survey data against a glossary of acceptable chemical names. Any chemical that does not match is referred to a chemist who determines whether the entry is a new chemical, a synonym for an acceptable chemical name, or a trade name that was improperly reported as a generic chemical.

Reporting the NOHSM Data

NIOSH is required by regulation to provide reports to each facility that it surveys. For NOHSM, NIOSH reports to each facility on the responses to the questionnaire and on the inventory of substances found on the property. The mine operator reviews this report for technical errors and for the inadvertent inclusion of confidential information. Later, when trade-name substances identified during the NOHSM surveys have been resolved into their associated chemical ingredients, the ingredients of trade-name substances that have not been declared trade secrets by their manufacturers will be available to the public.

After a commodity has been surveyed, NIOSH prepares a report that describes certain occupational health-related aspects of that commodity. The information is divided into several categories of potential exposures: chemicals with and without MSHA regulations and/or NIOSH-recommended exposure limits, trade-name products, physical agents, musculoskeletal overload conditions, and potential exposures from welding. The information is then presented in tabular form for each of these categories, giving the number of workers potentially exposed (both observed and predicted), the percentage of workers potentially exposed, the predicted annual usage of the product, the occupations of the workers exposed, and the locations in the facilities where exposure may occur.

NIOSH also plans to develop its capability of responding to special requests for analyses of NOHSM data. Experience with other data bases has shown that maximum speed and flexibility are essential elements of an efficient analytic and reporting system. Wherever possible, the system of NOHSM information will be combined with other mine-related information to provide a comprehensive reporting system.

Limitations of the NOHSM Data

The following limitations of the NOHSM data must be recognized: 1) The worksite data indicate only potential exposures. Thus, the agent may be observed at one or more sites in the facility, presenting the possibility that workers would be exposed to it. 2) Usage data are based on estimates provided by the mine management and should serve only as guides to the projected magnitude of usage rather than as precise information. Such estimates are expected to vary widely in accuracy. 3) The terminology relating to commodity, occupation, operation, and location was adapted directly from information that MSHA supplied to NIOSH. This terminology is not necessarily applied or accepted by others. 4) Mine operators have the right to specify which information they wish to have protected as trade secrets. For this reason, all NOHSM data that NIOSH reports to the public must be devoid of identifiable trade secrets. 5) NOHSM data are an indication of health-related conditions only at the time of the survey itself. No changes made by mine operators after the survey will be reflected in the data.

Observations from the First Segment

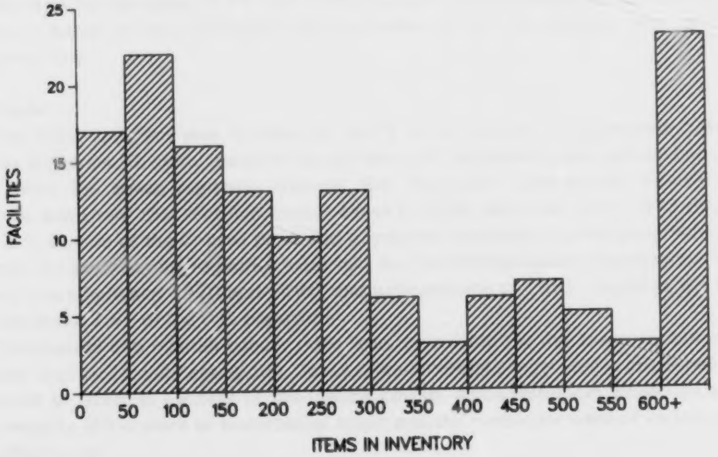
The first segment of NOHSM was concluded in September 1985. By October 28, 1985, 102 site reports either had been approved by the respective mine operators or were being reviewed by them. Although compilation of the findings is not yet complete, some descriptive information is available.

The number of trade-name products and chemical substances inventoried per site varies from fewer than 50 to more than 600 (Figure 1). Further analysis of the data may show this to be a function of the size of the site and/or the complexity of the operation carried out there. The current estimate of 35,000 products inventoried in this segment includes multiple listings of some products if they are found at more than one site; these duplicates are now being identified. An estimated 38% of the products inventoried were reported by mine management as not having been used during the previous 12 months. This high percentage was unexpected and is responsible, in part, for a longer survey time per site than had been predicted; the average survey time was 34 hours, compared with an expected time of 16 hours. Efforts to reduce the time required for surveys include the use of checklists, where feasible, and the omission of some zero-use products.

Follow-Up to NOHSM

Data derived from NOHSM will not completely meet the requirement of the Federal Mine Safety and Health Amendment Act of 1977 that levels of health-related agents be determined. Thus, once NIOSH learns from NOHSM which agents are present, follow-up work must begin that will identify monitoring strategies to yield the associated environmental levels. A complete study, of course, requires that the levels of all toxic agents be measured, including those that may not be visible, such as radon daughters and carbon monoxide.

FIGURE 1. Inventory of potentially hazardous products at mining facilities surveyed, United States, May 1984-September 1985



The Act also requires that information be gathered on a continuing basis. If NOHSM is to be a viable source of data in years to come, NIOSH will need an efficient mechanism for updating the NOHSM information, because it reflects conditions that existed only at the time of the survey.

Reference

1. CDC. Leading work-related diseases and injuries—United States. MMWR 1983;32:24-6, 32.

Trends in the Incidence of Endometrial and Ovarian Cancers

Marta L. Gwinn, M.D.
Division of Surveillance and Epidemiologic Studies
Epidemiology Program Office

Nancy C. Lee, M.D.
George L. Rubin, M.B.B.S.
Epidemiologic Studies Branch
Division of Reproductive Health
Center for Health Promotion and Education

Introduction

In the mid-1970s, a marked increase in the incidence of endometrial cancer occurred in the United States. After epidemiologic studies showed a strong association between the use of estrogen and the risk of endometrial cancer in postmenopausal women, the number of prescriptions for estrogen declined, and the number of new endometrial cancer cases decreased (*1,2*). In 1980, Jick et al. termed this episode "one of the largest epidemics of serious iatrogenic disease that has ever occurred in this country" and observed that ongoing surveillance could have detected the epidemic several years earlier (*1*).

Although no consistent association has been found between estrogen replacement therapy and risk of ovarian cancer (*3*), epidemiologic studies have suggested that the use of oral contraceptives reduces the risk of developing both ovarian and endometrial cancer (*4,5*). As women who have used oral contraceptives move into those age groups most likely to use estrogen replacement therapy—the age groups also at highest risk of developing endometrial or ovarian cancer—it is important to continue monitoring the incidence of these cancers.

For this report, we used data from the Surveillance, Epidemiology, and End Results (SEER) program (*6*) of the National Cancer Institute to calculate age-specific endometrial and ovarian cancer rates for the years 1973-1981. We also calculated corresponding rates for endometrial cancer based on the estimated true population at risk—i.e., women who have not had a hysterectomy.

Methods

The SEER program was founded in 1973 as a network of population-based regional cancer registries. To study trends in the incidence of endometrial and ovarian cancer, we used data from the seven registries (Detroit, San Francisco, Connecticut, Hawaii, Iowa, New Mexico, and Utah) that reported cancer cases to SEER each year from 1973 through 1981. The U.S. Bureau of the Census produced population estimates for the geographic areas covered by the participating registries especially for the SEER program, interpolating for the intercensal years between 1970 and 1980, and extrapolating to 1981. Together, the seven areas include about 7.5% of the U.S. population.

Endometrial and ovarian cancer rates were calculated for the seven registries combined, by year and by 10-year age group. Cases of endometrial cancer were considered to be those reported to SEER as cancers of the uterine corpus, since histologic diagnoses showed that the majority (83%) were of endometrial origin and the remainder were of various other or unspecified types.

No estimates of the national prevalence of hysterectomy in women beyond reproductive age have been reported since the early 1960s; therefore, we modified a method originally outlined by Lyon and Gardner to estimate the true population at risk of endometrial cancer (*7*). For a baseline, we used the proportion of women with an intact uterus in 1970, as estimated by Lyon and Gardner and refined by Marrett (*8*), interpolating to obtain estimates by single year of age. We then calculated hysterectomy incidence by single year of age for the years 1970-1980, using weighted data from the National Center for Health Statistics Hospital Discharge Survey and national population estimates (*9*). To estimate the population at risk of endometrial cancer by age in any given year, we calculated cumulative rates and applied them to SEER population data. These data, grouped into 10-year age intervals, provided the denominators for age-specific endometrial cancer rates based on the estimated true population at risk.

Results

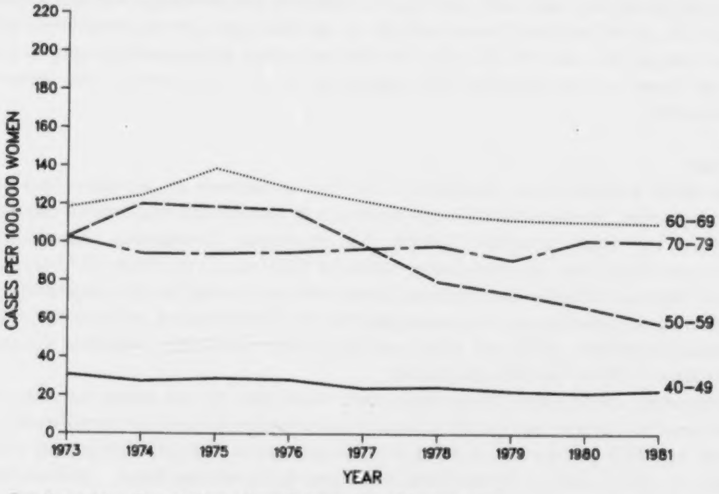
After reaching a peak in the mid-1970s, the incidence of endometrial cancer among women 50-69 years of age declined (Figure 1). During the same period, incidence of this cancer also declined among women younger than 50 years of age and remained essentially unchanged among women 70 years old and older (Table 1). Rates based on the estimated true population at risk (i.e., women with intact uteri) were higher, but overall trends were similar (Figure 2).

Between 1973 and 1981, the age-specific incidence of ovarian cancer changed very little (Table 2). No increasing trend was seen in any age group.

Discussion

In the mid-1970s, the recognition that estrogen replacement therapy was associated with endometrial cancer led to a change in physicians' prescribing practices; a reduction in endo-

FIGURE 1. Incidence of cancer of the uterine corpus, by age group and year, SEER (seven registries*), United States, 1973-1981



*Detroit, San Francisco, Connecticut, Hawaii, Iowa, New Mexico, Utah metrial cancer incidence followed within a few years. Recently, prescriptions for noncontraceptive estrogens have increased, but at lower doses and often in combination with progestins (*10*)—regimens thought to mitigate the associated risk of endometrial cancer (*11*). Ongoing surveillance will show whether the incidence of this cancer continues to decline.

Case-control studies have shown that a history of using oral contraceptives is associated with a reduced risk of developing endometrial and ovarian cancer. The growing proportion of "ever-users" of oral contraceptives in the age groups at highest risk should result in a decline in the incidence of both of these cancers. In 1981, some of the women who used oral contraceptives when they first became available in the 1960s had reached the 50- to 59-year age group; in contrast, women in the 60- to 69-year age group were unlikely to have ever used

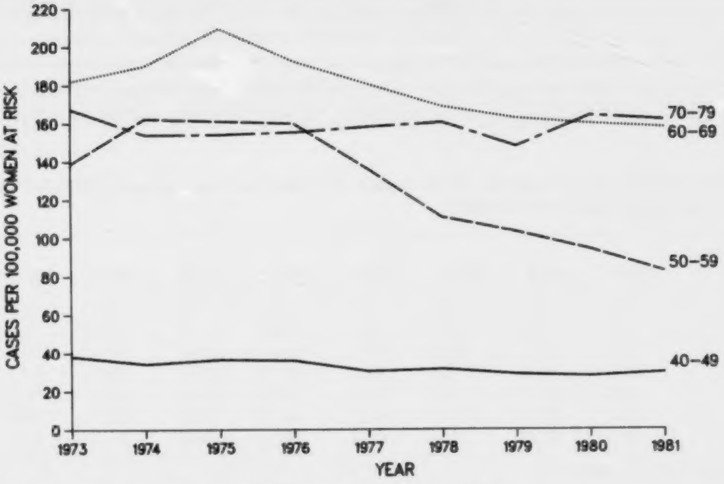
TABLE 1. Incidence* of cancer of the uterine corpus, by year and age group, SEER (seven registries†), United States, 1973-1981

Age group (years)	1973	1974	1975	1976	1977	1978	1979	1980	1981
20-29	1.2	1.3	0.7	0.8	0.9	0.9	0.8	0.7	0.4
30-39	6.7	5.5	6.1	5.6	5.2	5.9	4.8	4.4	5.6
40-49	30.7	27.3	28.9	28.3	23.8	24.6	22.4	21.6	23.0
50-59	102.9	120.1	118.6	116.9	98.6	79.7	73.6	66.5	58.1
60-69	118.4	124.5	138.5	128.5	121.7	115.2	112.0	111.1	110.3
70-79	102.6	94.1	94.1	94.9	96.8	98.5	91.1	101.5	100.7
≥80	46.9	57.1	65.9	70.6	66.2	83.5	66.3	66.2	66.1

*Cases per 100,000 women

†Detroit, San Francisco, Connecticut, Hawaii, Iowa, New Mexico, Utah

FIGURE 2. Incidence of cancer of the uterine corpus in women with intact uteri, by age group and year, SEER (seven registries*), United States, 1973-1981



*Detroit, San Francisco, Connecticut, Hawaii, Iowa, New Mexico, Utah oral contraceptives. Although the data presented indicate that endometrial cancer incidence continues to decline more rapidly among women in their 50s than among older women, this finding could well reflect different rates of exposure to noncontraceptive estrogens among women in these age groups.

The lack of national data on hysterectomy prevalence complicates the estimation of endometrial cancer rates based on the population truly at risk. The incidence of hysterectomy leveled off after increasing rapidly in the early 1970s (*12,13*); however, the age-specific prevalence of hysterectomy did not change enough between 1973 and 1981 to distort overall trends in endometrial cancer incidence.

Hysterectomy rates vary considerably by geographic region, being highest in the South and lowest in the Northeast (*13*). Thus, the proportion of the population at risk of endometrial cancer is likely to differ among the seven SEER areas included in this report. A survey in New York State (*14*) showed a lower prevalence of hysterectomy among women <40 years of age and among women 70-74 years of age than would be predicted by the model proposed by Lyon and Gardner. Estimates of hysterectomy prevalence would be preferable to those derived from an incidence-based model, which necessarily propagates errors in estimated hysterectomy incidence for any one year to all succeeding years.

Adequate data on prevalence of bilateral oophorectomy are not currently available. We expect that ovarian cancer rates based on the true population at risk would be higher than those shown; however, the differences should be somewhat less than those for endometrial cancer, since an estimated 30%-50% fewer women have undergone bilateral oophorectomy than hysterectomy (*13,14*).

Age-specific estimates of the prevalence of hysterectomy, as well as oophorectomy, will be available from data collected in the first phase of the National Health and Nutrition Examination Survey I—Epidemiologic Followup Survey (NHANES I—EFS), conducted by the National Center for Health Statistics between 1982 and 1984 (*15*). This survey combined information from questionnaires and medical records of women who were 25-74 years old when interviewed for NHANES I in 1971-1975.

The usefulness of data on incidence as well as on mortality for monitoring cancer trends has been well described (*16*). Although observed trends in cancer incidence may be influenced by changes in diagnostic procedures and case finding, they still may provide support for etiologic associations suggested by other studies and reflect changes in the prevalence of risk factors. Cohort analyses have helped isolate the effects of some of these factors in coun-

TABLE 2. Incidence[*] of cancer of the ovary, by year and age group, SEER (seven registries[†]), United States, 1973-1981

Age group (years)	1973	1974	1975	1976	1977	1978	1979	1980	1981
20-29	2.2	3.1	2.8	3.1	3.2	3.4	3.5	3.7	3.3
30-39	6.4	7.9	6.7	6.2	6.6	7.6	7.2	5.9	7.5
40-49	18.7	23.2	25.8	22.3	23.9	20.3	22.4	19.8	20.3
50-59	36.7	44.8	41.8	44.5	43.4	42.9	38.2	42.2	38.7
60-69	45.6	55.3	57.9	53.2	53.9	54.0	55.7	54.6	56.4
70-79	51.6	61.9	61.3	60.3	56.7	59.6	62.1	62.8	62.7
≥ 80	46.9	41.8	54.4	61.4	55.3	56.4	51.3	58.2	58.7

[*] Cases per 100,000 women

[†] Detroit, San Francisco, Connecticut, Hawaii, Iowa, New Mexico, Utah

tries where cancer surveillance has been under way for several decades (*17,18*). Such analyses may prove useful as surveillance under the SEER program continues.

References

1. Jick H, Walker AM, Rothman KJ. The epidemic of endometrial cancer: a commentary. Am J Public Health 1980;70:264-7.
2. Austin DF, Roe KM. The decreasing incidence of endometrial cancer: public health implications. Am J Public Health 1982;72:65-8.
3. Weiss NS, Lyon JL, Krishnamurthy S, Dietert SE, Liff JM, Daling JR. Noncontraceptive estrogen use and the occurrence of ovarian cancer. J Natl Cancer Inst 1982;68:95-8.
4. CDC. Cancer and Steroid Hormone Study. Oral contraceptive use and the risk of endometrial cancer. JAMA 1983;249:1600-4.
5. CDC. Cancer and Steroid Hormone Study. Oral contraceptive use and the risk of ovarian cancer. JAMA 1983;249:1596-9.
6. US Department of Health and Human Services. National Cancer Institute monograph 57. Surveillance, epidemiology, and end results: incidence and mortality data, 1973-1977. Bethesda, Maryland: US Department of Health and Human Services, 1981;NIH publication no. 81-2330.
7. Lyon JL, Gardner JW. The rising frequency of hysterectomy: its effect on uterine cancer rates. Am J Epidemiol 1977;105:439-43.
8. Marrett LD. Estimates of the true population at risk of uterine disease and an application to incidence data for cancer of the uterine corpus in Connecticut. Am J Epidemiol 1980;111:373-9.
9. US Bureau of the Census. Preliminary estimates of the population of the United States, by age, sex, and race: 1970 to 1981. Current population reports. Series P-25, no. 917. Washington, DC: US Bureau of the Census, July 1982.
10. Kennedy DL, Baum C, Forbes MB. Noncontraceptive estrogens and progestins: use patterns over time. Obstet Gynecol 1985;65:441-6.
11. Gambrell RD Jr, Bagnell CA, Greenblatt RB. Role of estrogens and progesterone in the etiology and prevention of endometrial cancer: review. Am J Obstet Gynecol 1983;146:696-707.
12. Perry BW. Time trends in hysterectomy 1970-1975. PAS Reporter 1976;14:1-6.
13. Dicker RC, Scally MJ, Greenspan JR, et al. Hysterectomy among women of reproductive age. JAMA 1982;248:323-7.
14. Howe HL. Age-specific hysterectomy and oophorectomy prevalence rates and the risks for cancer of the reproductive system. Am J Public Health 1984;74:560-3.
15. Cornoni-Huntley J, Barbano HE, Brody JA, et al. National Health and Nutrition Examination I—Epidemiologic Followup Survey. Public Health Rep 1983;96:245-51.
16. Devesa SS, Pollack ES, Young JL, Jr. Assessing the validity of observed cancer incidence trends. Am J Epidemiol 1984;119:274-89.
17. Ewertz M, Jensen OM. Trends in the incidence of cancer of the corpus uteri in Denmark, 1943-1980. Am J Epidemiol 1984;119:725-32.
18. Nischan P, Ebeling K. Oral contraceptives containing chlormadinone acetate and cancer incidence at selected sites in the German Democratic Republic—a correlation analysis. Int J Cancer 1984;34:671-4.

Ectopic Pregnancy in the United States, 1970-1983

Hani K. Atrash, M.D., M.P.H.
Joyce M. Hughes
Carol J.R. Hogue, Ph.D., M.P.H.
Division of Reproductive Health
Center for Health Promotion and Education

Introduction

In 1983, approximately 70,000 women were hospitalized for ectopic pregnancy in the United States (*1*). This represents a major public health problem in terms of fetal loss, the financial cost of caring for those women, and hundreds of thousands of workdays lost during hospitalization and rehabilitation of primarily healthy, young women. Furthermore, although it is a relatively rare complication of pregnancy (1.4% of all pregnancies in 1983), ectopic pregnancy accounted for 12.8% of all maternal deaths in the United States in 1983 (*1,2*). The second leading cause of pregnancy-related death (after toxemia), ectopic pregnancy was the leading cause of maternal deaths during the first trimester.

CDC has previously reported data on ectopic pregnancy for 1970-1980 (*3-5*). This report updates that information to include data for 1981-1983.

Methods

The estimated number of ectopic pregnancies was based on the National Hospital Discharge Survey (NHDS), conducted by the National Center for Health Statistics (NCHS). Each year, NCHS estimates the number of ectopic pregnancies nationwide by using a systematic sample of medical records (approximately 200,000) of patients discharged from about 400 nonfederal short-stay hospitals in the 50 states and the District of Columbia. The sample was stratified to represent all discharges from U.S. short-stay hospitals with respect to hospital size, type of ownership, and geographic location. For 1970-1978, abstracts with diagnosis code 631 according to the International Classification of Diseases, 8th Revision, Adapted for Use in the United States (ICDA-8) (*6*) were reported; for 1979-1983, abstracts with diagnosis code 633 according to the ICDA, 9th Revision (ICDA-9) (*7*) were reported. Data on deaths from ectopic pregnancy were based on U.S. vital statistics collected by NCHS. Ectopic pregnancy rates were calculated by dividing the estimated number of ectopic pregnancies by the reported number of pregnancies. Pregnancies were defined as the sum of live births, legally induced abortions, and ectopic pregnancies. The number of live births was obtained from NCHS, and the number of legally induced abortions was obtained from CDC's abortion surveillance data.

The geographic regions used in this report are those defined by the U.S. Bureau of the Census. Women were racially categorized as "white" or "black," with black including all races other than white. When information on race was not included in the medical records of women having ectopic pregnancies, we distributed the ectopic pregnancies according to the percentage of cases with known race; for 1981, NCHS distributed them according to race distributions in the geographic areas of the hospitals and on the basis of diagnoses and expected sources of payment as shown on medical records that included race. For the calculation of ectopic pregnancy rates, women were grouped into three age categories: 15-24, 25-34, and 35-44 years. For ectopic pregnancy mortality analysis, women were grouped into six age categories: 15-19, 20-24, 25-29, 30-34, 35-39, and 40-44 years. Because of small numbers of deaths, women 35-44 years old were combined into one age group for comparison purposes. In previous publications CDC has reported mortality due to ectopic pregnancy as deaths per 1,000 cases; however, because mortality rates have declined to less than one death per 1,000 cases of ectopic pregnancies, in this publication mortality is reported as deaths per 10,000 cases. Estimates of the number of ectopic pregnancies have been rounded to the nearest 100. The rounding and the redistribution of cases in which race is unknown sometimes cause the sum of individual numbers not to equal the total. Rates were calculated from the unrounded estimates.

Results

For the period 1970-1983, an estimated 563,100 ectopic pregnancies occurred among women 15-44 years old in the United States, with an overall rate of 9.2 per 1,000 reported pregnancies (Table 1, Figure 1). Except for 1982, the number of ectopic pregnancies increased each year from 1970 through 1983.

Between 1970 and 1983, the total number of ectopic pregnancies increased 3.9-fold, from 17,800 to 69,600 per year. For white women, the number increased fourfold, from 12,900 in 1970 to 51,800 in 1983; for black women, the number increased 3.6-fold, from 4,900 in 1970 to 17,800 in 1983. For all women combined, the rate increased 3.1-fold, from 4.5 in 1970 to 14.0 in 1983; rates increased 3.4-fold and 2.2-fold for white and black women, respectively (Table 1).

For the period 1970-1983, the risk of ectopic pregnancy increased with age and was highest for women 35-44 years old. White women 35-44 years of age had a threefold higher risk than white women 15-24 years old, whereas black women 35-44 years old had a 4.3-fold higher risk than black women 15-24 years old (Table 2, Figure 2).

TABLE 1. Number and rate of ectopic pregnancies for females ages 15-44, by race* and year, United States, 1970-1983

Year	Number†			Rate§		
	White	Black	Total	White	Black	Total
1970	12,900	4,900	17,800	4.0	7.1	4.5
1971	13,900	5,300	19,300	4.2	7.3	4.8
1972	17,300	7,200	24,500	5.5	9.8	6.3
1973	16,600	9,000	25,600	5.5	11.9	6.8
1974	19,800	6,600	26,400	6.3	8.1	6.7
1975	21,700	8,900	30,500	6.9	10.2	7.6
1976	24,900	9,700	34,600	7.7	10.4	8.3
1977	28,900	11,900	40,700	8.4	11.8	9.2
1978	29,500	12,800	42,400	8.5	12.3	9.4
1979	35,300	14,600	49,900	9.5	13.4	10.4
1980	36,500	15,600	52,200	9.5	14.0	10.5
1981	48,400	19,600	68,000	12.5	17.6	13.6
1982	49,400	12,400	61,800	12.7	10.9	12.3
1983	51,800	17,800	69,600	13.6	15.5	14.0
Total	**406,800**	**156,200**	**563,100**	**8.4**	**11.9**	**9.2**

*Race "unknown" redistributed according to the percentage of race known

†Rounded to nearest hundred; estimates based on <10,000 ectopic pregnancies should be viewed with caution

§Rate per 1,000 reported pregnancies (live births, legally induced abortions, and ectopic pregnancies)

Overall, black women had a 1.4-fold higher risk of ectopic pregnancy than white women. The increased risk existed among all age groups and in all regions except in the West, where the risk was slightly higher for white women (Table 2). Black women 35-44 years old had the highest risk of ectopic pregnancy. Their risk was five times higher than that of white women 15-24 years old, the group with the lowest rate.

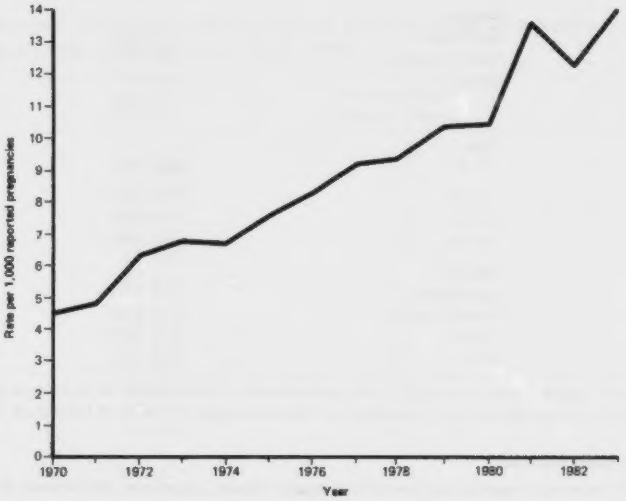
The overall rates of ectopic pregnancy were similar in the four geographic regions (Table 2). However, the race- and region-specific rates showed some variability. For white women the highest rates were in the West and the lowest in the South, whereas for black women the highest rates were in the North Central region and the lowest in the West (Table 2).

The average length of stay among women hospitalized for ectopic pregnancy decreased from 7.2 days in 1970 to 4.8 days in 1983. Despite this decline, the estimated total number of hospitalization days for women with ectopic pregnancy almost tripled from 1970 to 1983 because of the increased number of ectopic pregnancies (Table 3).

In the period 1970-1983, 644 women in the United States died from ectopic pregnancy, making the death rate 11.4 per 10,000 ectopic pregnancies. The risk of death decreased 6.7-fold, from 35.5 per 10,000 ectopic pregnancies in 1970 to 5.3 in 1983 (Figure 3, Table 4). Analysis by race showed similar declines—from 21.7 to 3.3 deaths per 10,000 ectopic pregnancies (6.6-fold) for white women, and from 72.1 to 11.2 deaths per 10,000 (6.4-fold) for black women (Table 4).

For the period 1970-1983, black women were at higher risk of death from ectopic pregnancy than white women, independent of age or geographic location (Table 5). For white women, the highest risk of death was among those 15-19 years old. The risk decreased with age, with women 40-44 years old having the lowest risk. For black women, the lowest risk of death was among those 25-29 years old; the risk was higher for younger and for older women (Table 5, Figure 4).

FIGURE 1. Ectopic pregnancy rates for females ages 15-44, by year, United States, 1970-1983



Discussion

The numbers and rates of ectopic pregnancy have increased every year from 1970 through 1983, except for 1982. Analysis revealed that the number of ectopic pregnancies in 1981 was much higher than expected on the basis of rates of increase during the period 1970-1980 (Figure 1). This resulted in an apparent decrease in the estimated number of ectopic pregnancies for 1982; however, that number was within expectations based on the rate of increase during the preceding 12 years. We cannot explain the disproportionate increase in the number of ectopic pregnancies in 1981, particularly among blacks.

TABLE 2. Number and rate of ectopic pregnancies, by race, age, and geographic region, United States, 1970-1983

Race*	Age/Region	Number†	Rate§
White	**Age:**		
	15-44	406,800	8.4
	15-24	133,800	5.3
	25-34	232,000	11.3
	35-44	41,000	15.9
	Region:		
	Northeast	86,400	8.2
	North Central	108,700	8.5
	South	107,800	7.6
	West	106,200	9.8
Black	**Age:**		
	15-44	156,200	11.9
	15-24	51,400	6.4
	25-34	83,900	19.0
	35-44	20,900	27.7
	Region:		
	Northeast	37,000	12.5
	North Central	34,700	14.3
	South	64,400	11.4
	West	17,900	9.2
All races	**Age:**		
	15-44	563,100	9.2
	15-24	185,200	5.6
	25-34	316,000	12.7
	35-44	61,900	18.6
	Region:		
	Northeast	123,300	9.2
	North Central	143,500	9.4
	South	172,200	8.7
	West	124,100	9.7

*Race "unknown" redistributed according to the percentage of race known for each age group and each geographic region. Redistribution and rounding sometimes cause the sum of individual numbers not to equal the total.

†Rounded to nearest hundred

§Rate per 1,000 reported pregnancies (live births, legally induced abortions, and ectopic pregnancies)

FIGURE 2. Ectopic pregnancy rates, by race and age group, United States, 1970-1983

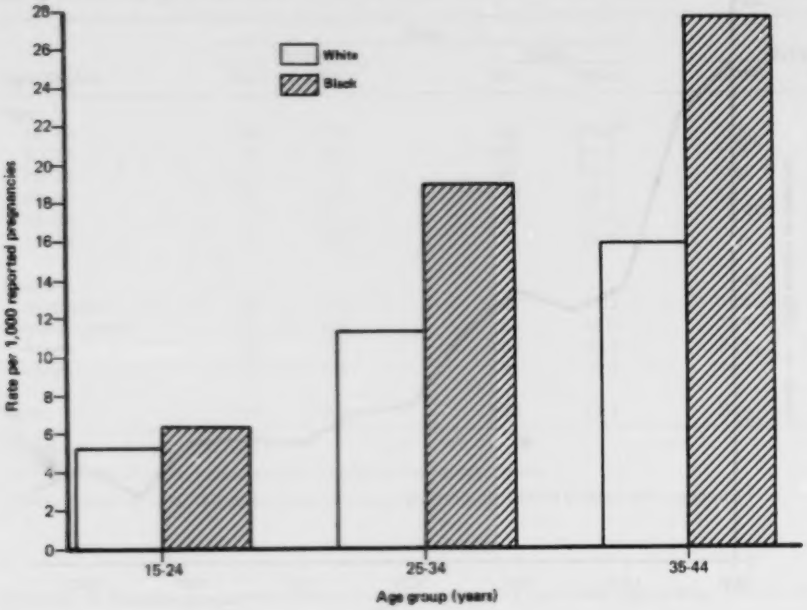


TABLE 3. Number of ectopic pregnancies and average length of hospital stay for females ages 15-44, by year, United States, 1970-1983

Year	Number	Average length of stay (days)	Total person-days hospitalized
1970	17,800	7.2	127,800
1971	19,300	7.1	136,700
1972	24,500	7.1	173,800
1973	25,600	7.4	189,200
1974	26,400	6.8	179,200
1975	30,500	6.1	186,200
1976	34,600	6.4	221,600
1977	40,700	5.8	236,200
1978	42,400	5.9	249,900
1979	49,900	5.9	294,300
1980	52,200	5.8	302,600
1981	68,000	5.5	374,000
1982	61,800	5.2	321,400
1983	69,600	4.8	334,100
Total	**563,100**	**5.9**	**3,327,000**

FIGURE 3. Ectopic pregnancy death-to-case rates, by year, United States, 1970-1983

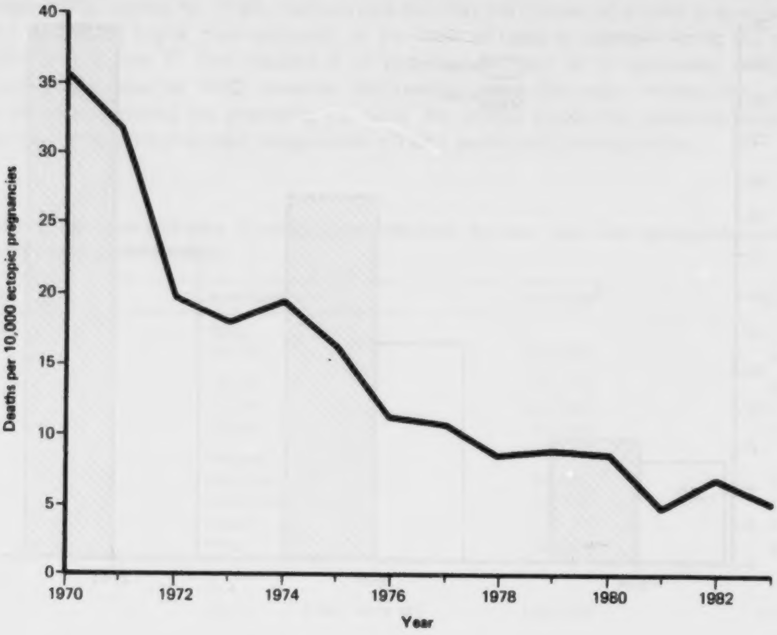


TABLE 4. Number and rate of deaths due to ectopic pregnancy, by race and year, United States, 1970-1983

Year	Number			Rate*		
	White	Black	Total	White	Black	Total
1970	28	35	63	21.7	72.1	35.5
1971	21	40	61	15.1	74.9	31.7
1972	28	20	48	16.2	27.7	19.6
1973	25	21	46	15.1	23.4	18.0
1974	20	31	51	10.1	47.0	19.4
1975	19	31	50	8.8	34.9	16.4
1976	11	28	39	4.4	28.7	11.3
1977	15	29	44	5.2	24.5	10.8
1978	13	24	37	4.4	18.7	8.7
1979	20	25	45	5.7	17.2	9.0
1980	22	24	46	6.0	15.4	8.8
1981	15	19	34	3.1	9.7	5.0
1982	19	24	43	3.8	19.3	7.0
1983	17	20	37	3.3	11.2	5.3
Total	**273**	**371**	**644**	**6.7**	**23.7**	**11.4**

*Deaths from ectopic pregnancy per 10,000 ectopic pregnancies. To compare these rates with previously reported rates per 1,000 ectopic pregnancies, divide the above rate by 10.

TABLE 5. Death-to-case rates for women with ectopic pregnancies, by race, age, and geographic region, United States, 1970-1983

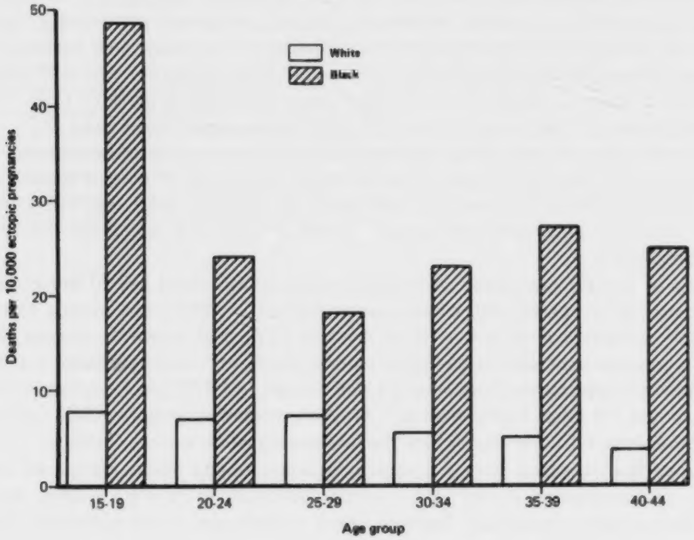
	Race					
	White		Black		Total	
Age/Region	No.[*]	Rate[†]	No.	Rate	No.	Rate
Age:						
15-19	22	7.9	43	48.8[§]	65	17.8
20-24	75	7.1	103	24.2	178	12.0
25-29	107	7.4	91	18.2	198	10.2
30-34	49	5.6	78	22.9	127	10.4
35-39	17	5.1	45	27.3	62	12.5
40-44	3	3.8[§]	11	24.9[§]	14	11.3
Region:						
Northeast	62	7.2	86	23.3	148	12.0
North Central	63	5.8	87	25.0	150	10.5
South	80	7.4	163	25.3	243	14.1
West	68	6.4	35	19.6	103	8.3
Total	**273**	**6.7**	**371**	**23.7**	**644**	**11.4**

[*] Deaths from ectopic pregnancy

[†] Deaths from ectopic pregnancy per 10,000 ectopic pregnancies

[§] Rates based on < 10,000 ectopic pregnancies and thus should be viewed with caution

FIGURE 4. Ectopic pregnancy death-to-case rates, by race and age group, United States, 1970-1983



Several hypotheses have been published to explain the occurrence of ectopic pregnancy. Weckstein (*8*), in 1985, grouped causes of tubal pregnancies into two categories: 1) maternal factors, which delay or prevent a fertilized egg from passing into the uterine cavity, and 2) fetal factors, which are inherent in the embryo. Maternal factors include pelvic inflammatory disease (PID), congenital tubal malformation, tubal ligation, and menstrual regurgitation of a fertilized ovum. Fetal factors include embryonic malformations and chromosomal anomalies. Of all these factors, PID is probably the most commonly agreed upon explanation for the ever-increasing incidence of ectopic pregnancy; however, the magnitude of PID's contribution to this increasing incidence is not agreed upon. Breen (*9*) reported that 92% of 684 ectopic pregnancies could be explained by PID (42% of the patients gave a history of PID; 50% gave a history of antibiotic therapy, implying PID). But Niles and Clark (*10*), by pathologic analysis of 436 ectopic pregnancies, found evidence of PID in only 40% of the cases. They concluded that no evidence of PID in 60% of the cases suggests a functional pathogenesis. The increase in PID that has occurred in the United States during the past two decades is responsible for at least part of the increased incidence of ectopic pregnancy (*11*).

Another suggested contributing factor to the increasing incidence of ectopic pregnancy is the increasing proportion of women who are discontinuing the use of oral contraceptives, which have been shown to protect against ectopic pregnancy by depressing ovulation (*12*). NCHS reported that the proportion of women using oral contraception among currently married women dropped from 25.1% in 1973 to 13.5% in 1982 (*13*); however, this trend is not likely to explain the increase in ectopic pregnancies. NCHS also reported that the proportion of currently married women who are surgically sterilized increased from 22.9% to 38.9% during the same period, whereas the proportion of those using other methods of contraception (e.g., intrauterine devices, condoms, diaphragms, foam, abstinence, withdrawal, or douching) essentially did not change (21.5% in 1973 and 21.8% in 1982) (*13*). Women are apparently giving up oral contraception for surgical sterilization, known to have a lower failure rate and to be associated with a very low incidence of ectopic pregnancies. On the basis of 1) the reported incidence of ectopic pregnancy following tubal sterilization and 2) the reported numbers of sterilizations in the United States, Barnes et al. concluded that tubal sterilization in the United States might precede one to two ectopic pregnancies per 100,000 females ages 15-44, and it does not appear to be a major cause of ectopic pregnancy (*14*). The rate of ectopic pregnancy in 1983 was 126 per 100,000 females ages 15-44 years (*1*).

Improved diagnostic technology (particularly the widespread use of laparoscopy and ultrasonography) may have contributed to an increased recognition of ectopic pregnancies. This, in turn, results in earlier diagnoses and management, leading to improved outcomes. In addition, the improved diagnostic technology partially explains the decreased mortality due to ectopic pregnancy.

Results of ectopic pregnancy surveillance show an increased risk of ectopic pregnancy among black as compared with white women (except in 1982), and among older as compared with younger women in the 15- to 44-year age group. However, whereas the rate of ectopic pregnancy continues to be higher among blacks than among whites, the rate among white women is approaching that among black women. In 1970, black women had an ectopic pregnancy rate 1.8 times higher than that of white women, whereas in 1983 the rate among black women was 1.1 times higher than the rate among white women (Table 1).

Although the increased rate of ectopic pregnancy among blacks compared with whites seems to be disappearing, in 1983 black women continued to have a 3.5-times-higher risk of death from ectopic pregnancy. Teenage black women are at an extremely high risk of death—6.2 times higher than white teenagers. Black teenagers are at a 10-times-higher risk of death than white women 35-44 years of age, the group with the lowest risk of death.

Deaths due to ectopic pregnancy can be related to the timing and quality of prenatal care. Early prenatal care provides for the timely diagnosis and management of ectopic pregnancy. Fewer blacks and younger women than whites and older women are reported to have prenatal care and to start such care during the first trimester (*15*). This may help explain the higher mortality rate among blacks and younger women than among whites and older women.

Finally, because the NHDS is a sample survey that does not include medical records of patients discharged from federal hospitals, and because some ectopic pregnancies are known to resolve spontaneously and therefore remain undiagnosed, the reported number of ectopic pregnancies undoubtedly is an underestimate of the true number. Furthermore, our estimate of reported pregnancies is an underestimate because it does not include spontaneous abortions and because the number of legally induced abortions reported to CDC is consistently lower than the number that the Alan Guttmacher Institute obtains by direct survey of legal abortion providers. Our estimates of ectopic pregnancy death rates, therefore, are somewhat high.

References

1. CDC. Ectopic pregnancy—United States, 1981-1983. MMWR 1986;35:289-91.
2. National Center for Health Statistics. Monthly Vital Statistics 1985;34(6), suppl.
3. CDC. Ectopic pregnancy surveillance, 1970-1978. Atlanta, Georgia: CDC, July 1982.
4. MacKay HT, Hughes JM, Hogue CR. Ectopic pregnancy in the United States, 1979-1980. In: CDC surveillance summaries. Atlanta, Georgia: CDC, 1984;33(2SS):1SS-7SS.
5. Rubin GL, Peterson HB, Dorfman SF, et al. Ectopic pregnancy in the United States, 1970 through 1978. JAMA 1983;249:1725-9.
6. International Classification of Diseases, 8th Revision, Adapted for Use in the United States. Washington, DC: National Center for Health Statistics, 1967.
7. International Classification of Diseases, 9th Revision, Adapted for Use in the United States. Ann Arbor, Michigan: National Center for Health Statistics, 1978.
8. Weckstein LN. Current perspective on ectopic pregnancy. Obstet Gynecol Surv 1985;40:259-72.
9. Breen JL. A 21-year survey of 654 ectopic pregnancies. Am J Obstet Gynecol 1970;106:1004-19.
10. Niles JH, Clark JFJ. Pathogenesis of tubal pregnancy. Am J Obstet Gynecol 1969;105:1230-4.
11. Washington AE, Cates W Jr, Zaidi AK. Hospitalizations for pelvic inflammatory disease: epidemiology and trends in the United States, 1975 to 1981. JAMA 1984;251:2529-33.
12. Ory HW, et al. (Women's Health Study). Ectopic pregnancy and intrauterine contraceptive devices: new perspectives. Obstet Gynecol 1981;57:137-44.
13. National Center for Health Statistics. Advancedata No. 102. Hyattsville, Maryland: NCHS, 1984.
14. Barnes BB, Wennberg CN, Barnes BA. Ectopic pregnancy: incidence and review of determinant factors. Obstet Gynecol Surv 1983;38:345-56.
15. National Center for Health Statistics. Health United States 1984. Hyattsville, Maryland: NCHS, 1984; DHHS publication no. (PHS) 85-1232.

Copies can be purchased from:
Superintendent of Documents
U.S. Government Printing Office
Washington, D.C. 20402
Telephone: (202) 783-3238

State and Territorial Epidemiologists and State Laboratory Directors

The contributions of the State and Territorial Epidemiologists and the State Laboratory Directors to this report are gratefully acknowledged. The persons listed were in the positions shown as of August 1, 1986.

State	Epidemiologists	Laboratory Directors
Alabama	Wallace E. Birch, DVM	James L. Holston, Jr., DrPH
Alaska	John P. Middaugh, MD	Vacant
Arizona	Glyn G. Caldwell, MD	Jon M. Counts, DrPH
Arkansas	Thomas C. McChesney, DVM	Robert L. Horn
California	James Chin, MD, MPH	G. W. Fuhs, DrSciNat
Colorado	Stanley W. Ferguson, PhD	Ronald L. Cada, DrPH
Connecticut	James L. Hadler, MD, MPH	Jesse Tucker, PhD
Delaware	Paul R. Silverman, DrPH	Mahadeo P. Verma, PhD
District of Columbia	Martin E. Levy, MD, MPH	James B. Thomas, ScD
Florida	Michael H. Wilder, MD, Acting	Eldert C. Hartwig, ScD
Georgia	R. Keith Sikes, DVM	Frank M. Rumph, MD
Hawaii	Arthur P. Liang, MD, MPH	Glenn Kobayashi, Acting
Idaho	Charles D. Brokopp, DrPH	Darrell W. Brock, DrPH
Illinois	Byron J. Francis, MD	Robert Coe, Acting
Indiana	Charles L. Barrett, MD	T. L. Eddleman
Iowa	Laverne A. Wintermeyer, MD	W. J. Hausler, Jr., PhD
Kansas	Robert French, Acting	Roger H. Carlson, PhD
Kentucky	Joseph W. Skaggs, DVM, MPH, Acting	B. F. Brown, MD
Louisiana	Joyce B. Mathison, MD	Henry Bradford, PhD
Maine	Kathleen F. Gensheimer, MD	Philip W. Haines, DrPH
Maryland	Ebenezer Israel, MD	J. Mehsen Joseph, PhD
Massachusetts	George F. Grady, MD	Harvey George, PhD
Michigan	Kenneth R. Wilcox, Jr., MD	George R. Anderson, DVM
Minnesota	Michael T. Osterholm, PhD, MPH	Robert W. Hiller
Mississippi	Fred E. Thompson, MD	R. H. Andrews, MPH
Missouri	H. Denny Donnell, Jr., MD, MPH	Elmer R. Spurrier, DrPH
Montana	Judith K. Gedrose, MN	Douglas Abbott, PhD
Nebraska	Paul A. Stoesz, MD	John Blosser
Nevada	George E. Reynolds, MD, Acting	George E. Reynolds, MD
New Hampshire	Eugene Schwartz, MD	Veronica C. Malmberg
New Jersey	William E. Parkin, DVM	Shaheidy I. Shaheid, PhD
New Mexico	Harry F. Hull, MD	Loris W. Hughes, PhD
New York State	Dale L. Morse, MD	Herbert W. Dickerman, MD, PhD
New York City	Stephen Schultz, MD	Paul S. Maye, DSc
North Carolina	J. N. MacCormack, MD, MPH	Mildred A. Kerbaugh
North Dakota	James L. Pearson, DrPH	Jane S. Robb, Acting
Ohio	Thomas J. Halpin, MD, MPH	Gary D. Davidson, DrPH
Oklahoma	Gregory R. Istre, MD	Garry L. McKee, PhD
Oregon	Laurence R. Foster, MD, MPH	Michael R. Skeels, PhD
Pennsylvania	Ernest J. Witte, VMD, MPH	Vern Pidcoe, DrPH
Rhode Island	Richard A. Keenlyside, MD	Raymond G. Lundgren, Jr., PhD
South Carolina	Richard L. Parker, DVM	Arthur F. DiSalvo, MD
South Dakota	Kenneth A. Senger	A. Richard Melton, DrPH
Tennessee	Robert H. Hutcheson, MD	Michael W. Kimberly, DrPH
Texas	Charles E. Alexander, MD	Charles E. Sweet, DrPH
Utah	Craig R. Nichols, MPA	Francis M. Urry, PhD
Vermont	Richard L. Vogt, MD	Katherine A. Kelley, DrPH
Virginia	Grayson B. Miller, Jr., MD	Frank W. Lambert, Jr., PhD
Washington	John M. Kobayashi, MD	Jack Allard, PhD
West Virginia	Loretta E. Haddy, MS	John W. Brough, DrPH
Wisconsin	Jeffrey P. Davis, MD	Ronald H. Laessig, PhD
Wyoming	Harry C. Crawford, MD	Donald T. Lee, PhD
Guam	Robert L. Haddock, DVM	Angelina Roman, Acting
Federated States of Micronesia	Eliuel K. Pretrick, MO	Vacant
Marshall Islands	Isao Kisino	Vacant
Northern Mariana Is.*	Jose T. Villagomez, MO	Vacant
American Samoa	Julia L. Lyons, MD, MPH	Vacant
Palau*	Anthony H. Polloi, MO, Acting	Vacant
Puerto Rico	Jose G. Rigau, MD	Raul Baco, MD
Virgin Islands	John N. Lewis, MD	Norbert Mantor

---

*Formerly Trust Territory of the Pacific Islands

☆U.S. Government Printing Office: 1986-746-149/40022 Region IV

UNITED STATES GOVERNMENT PRINTING OFFICE
SUPERINTENDENT OF DOCUMENTS
Washington, D.C. 20402

OFFICIAL BUSINESS
Penalty for Private Use, $300

BULK RATE
POSTAGE & FEES PAID
GPO
Permit No. G-26

MMWR NEWAROOON DR 376 F 1 *
NEWARK PUBLIC LIBR *
5 WASHINGTON ST *
NEWARK NJ 07101 *
*

HHS Publication No. (CDC) 86-8017

Redistribution using indicia is illegal.